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NEWS 4	Feb 01	DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS 5	Feb 19	Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 6	Mar 08	Gene Names now available in BIOSIS
NEWS 7	Mar 22	TOXLIT no longer available
NEWS 8	Mar 22	TRCTHERMO no longer available
NEWS 9	Mar 28	US Provisional Priorities searched with P in CA/CAplus and USPATFULL
NEWS 10	Mar 28	LIPINSKI/CALC added for property searching in REGISTRY
NEWS 11	Apr 02	PAPERCHEM no longer available on STN. Use PAPERCHEM2 instead.
NEWS 12	Apr 08	"Ask CAS" for self-help around the clock
NEWS 13	Apr 09	BEILSTEIN: Reload and Implementation of a New Subject Area
NEWS 14	Apr 09	ZDB will be removed from STN
NEWS 15	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS 16	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 17	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 18	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS 19	Jun 03	New e-mail delivery for search results now available
NEWS 20	Jun 10	MEDLINE Reload
NEWS 21	Jun 10	PCTFULL has been reloaded
NEWS 22	Jul 02	FOREGE no longer contains STANDARDS file segment

NEWS EXPRESS	February 1 CURRENT WINDOWS VERSION IS V6.0d, CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP), AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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FILE 'HOME' ENTERED AT 11:40:44 ON 11 JUL 2002

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=> file biosis medline agricola embase caba wpids  japio biotechds lifesci caplus
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COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
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=> e leonard john p/au
E1          79     LEONARD JOHN M/AU
E2           3     LEONARD JOHN N/AU
E3         106 --> LEONARD JOHN P/AU
E4           1     LEONARD JOHN PATRICK/AU
E5           1     LEONARD JOHN PAUL/AU
E6           2     LEONARD JOHN R/AU
E7           3     LEONARD JOHN S/AU
E8           1     LEONARD JOHN T/AU
E9           1     LEONARD JOHN W JR/AU
E10          1     LEONARD JOHNATAN N/AU
E11          1     LEONARD JOHNSON F/AU
E12          4     LEONARD JON/AU

=> s e3-e5
L1         108 ("LEONARD JOHN P"/AU OR "LEONARD JOHN PATRICK"/AU OR "LEONARD
               JOHN PAUL"/AU)

=> e leonard j p/au
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E1            1      LEONARD J M R M/AU
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E6            3      LEONARD J R 3RD/AU
E7            3      LEONARD J R III/AU
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E9            2      LEONARD J S JR/AU
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=> s e3-e4
L2          359 ("LEONARD J P"/AU OR "LEONARD J P *"/AU)

=> e goldman samuel/au
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E4            6      GOLDMAN SAMUEL C/AU
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=> s e2-e9
L3           78 ("GOLDMAN SAM"/AU OR "GOLDMAN SAMUEL"/AU OR "GOLDMAN SAMUEL
              C"/AU OR "GOLDMAN SAMUEL D"/AU OR "GOLDMAN SAMUEL J"/AU OR "GOLD
              MAN SAMUEL JAY"/AU OR "GOLDMAN SAMUEL L"/AU OR "GOLDMAN SAMUEL
              M"/AU)

=> e goldman s/au
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E4          245      GOLDMAN S A/AU
E5            3      GOLDMAN S A */AU
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E7           20      GOLDMAN S C/AU
E8            2      GOLDMAN S D/AU
E9           24      GOLDMAN S E/AU
E10          15      GOLDMAN S F/AU
E11           9      GOLDMAN S G/AU
E12          11      GOLDMAN S H/AU

=> s e3
L4         1458 "GOLDMAN S"/AU

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E7            1      OHARA RIKURO/AU
E8            1      OHARA RURI/AU
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E9            3     OHARA RYO/AU
E10          13     OHARA RYOICHI/AU
E11           1     OHARA RYOUICHI/AU
E12           2     OHARA RYUICHI/AU

=> e o'hara richard/au
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off or masking.

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E3           70 --> OHARA R/AU
E4            1     OHARA R B/AU
E5            1     OHARA R D/AU
E6            4     OHARA R J/AU
E7            1     OHARA R P/AU
E8            1     OHARA R S/AU
E9            1     OHARA REIJI/AU
E10          19     OHARA REIKO/AU
E11           1     OHARA RIE/AU
E12           1     OHARA RIEKO/AU

=> s e3
L5           70 "OHARA R"/AU

=> s l1-l5
L6         2059 (L1 OR L2 OR L3 OR L4 OR L5)

=> s l6 and (IL-12 or RA or arthritis)
L7          109 L6 AND (IL-12 OR RA OR ARTHRITIS)

=> s l7 and (antibod? or antagonist?)
L8           43 L7 AND (ANTIBOD? OR ANTAGONIST?)

=> dup rem l8
PROCESSING COMPLETED FOR L8
L9            20 DUP REM L8 (23 DUPLICATES REMOVED)

=> d bib ab 1-20

L9   ANSWER 1 OF 20  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE
     1
AN   2002:166945  BIOSIS
DN   PREV200200166945
TI   Use of IL-12 and IL-12
     antagonists in the treatment of autoimmune diseases.
AU   Leonard, John (1); Goldman, Samuel; O'Hara, Richard, Jr.
CS   (1) Auburn, NH USA
```

ASSIGNEE: Genetics Institute, Inc.
PI US 6338848 January 15, 2002
SO Official Gazette of the United States Patent and Trademark Office Patents, (Jan. 15, 2002) Vol. 1254, No. 3, pp. No Pagination. http://www.uspto.gov/web/menu/patdata.html. e-file. ISSN: 0098-1133.
DT Patent
LA English
AB Method of treating autoimmune conditions are disclosed comprising administering to a mammalian subject **IL-12** or an **IL-12 antagonist**. In certain preferred embodiments the autoimmune condition is one which is promoted by an increase in levels of IFN-gamma or TNF-alpha. Suitable conditions for treatment include multiple sclerosis, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent diabetes melitis and autoimmune inflammatory eye disease.

L9 ANSWER 2 OF 20 WPIDS (C) 2002 THOMSON DERWENT
AN 2002-147853 [19] WPIDS
DNC C2002-045892
TI Composition for modulating immune response, comprises a spore system having a spore and polypeptide, carbohydrate or nucleotide sequence having anti-pathogenic activity.
DC B04 D16
IN **GOLDMAN**, **S**; LATHROP, S J; LONGCHAMP, P F; WHALEN, R G
PA (MAXY-N) MAXYGEN INC
CYC 96
PI WO 2002000232 A2 20020103 (200219)* EN 137p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001073009 A 20020108 (200235)
ADT WO 2002000232 A2 WO 2001-US20372 20010626; AU 2001073009 A AU 2001-73009 20010626
FDT AU 2001073009 A Based on WO 200200232
PRAI US 2000-214161P 20000626
AB WO 200200232 A UPAB: 20020321
NOVELTY - A composition (I), comprising a spore system (II) having a spore and a peptide, polypeptide, protein, carbohydrate or nucleotide sequence having anti-pathogenic activity displayed on, bound to or contained within, the spore, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) releasing a spore system of interest, comprising:
(a) transforming a cell capable of sporulation with an exogenous nucleic acid;
(b) inducing sporulation of the cell, where at least one spore system is produced; and
(c) lysing the cell to release the spore system;
(2) displaying a polypeptide at one or more sites of interest on a surface of a spore, comprising:
(a) transforming a cell capable of sporulation with a recombinant nucleic acid vector, comprising a nucleic acid encoding a polypeptide fused in frame to a nucleic acid encoding a spore protein; and
(b) expressing a fusion protein comprising the polypeptide and the spore coat protein so that the fusion protein is attached to the spore coat of the spore at one or more site of interest on the spore surface;
(3) a detection system (DS) comprising (II) which comprises a moiety

that provides a detectable signal and a polypeptide capable of capturing a detectable compound;

(4) delivery of a polypeptide of interest, comprising:

(a) transforming a cell that is capable of sporulating with a nucleic acid encoding the polypeptide;

(b) inducing sporulation of the cell to form a spore; and

(c) delivering the spore to a site of interest;

(5) modulation of an adjuvant effect in an organism, comprising:

(a) generating a non-viable spore, having an adjuvant effect;

(b) isolating the spore; and

(c) contacting the organism with the spore and a nucleic acid, polypeptide, or peptide; and

(6) enhancing (M1) an immune response to an immunogenic polypeptide or peptide in a subject, comprising administering (I).

ACTIVITY - Antibacterial; virucide; anti-HIV (human immunodeficiency virus); cytostatic; neuroprotective; nootropic; hepatotropic; antipyretic; antiinflammatory; antirheumatic; antiarthritic; antidiabetic; immunosuppressive; antipsoriatic; antiallergic; antiasthmatic.

MECHANISM OF ACTION - Modulator of immune response (claimed); vaccine.

Spores from Bacillus subtilis were tested to determine if the spores had an adjuvant effect. The specific immunological response of mice to spores and V-antigen mixed together was compared to the specific immunological response of mice to the V-antigen protein alone. 1 micro g, 0.5 micro g or 0.25 micro g of purified recombinant V-antigen was mixed with 5 multiply 108 non-recombinant B. subtilis spores or used alone. The three V-antigen protein/spore mixtures and three amounts of V-antigen protein were injected intraperitoneally into separate groups of mice, at days 1, 21 and 35. Mice were bled on days 10, 21 and 45. Serum was analyzed for specific and V-antigen immunoglobulins by an indirect enzyme linked immunosorbent assay (ELISA) using standard procedures. The presence of spores in the inoculum increased the **antibody** titer between 10-fold and 1000-fold, depending on the amount of protein inoculated. The data suggested that spores act to augment a specific immune response to immunogenic polypeptide, such as V-antigen protein.

USE - (I) is useful for modulating (producing or enhancing) an immune response of an organism, and for generating a desired product. DS is useful for detecting a compound. (All claimed). The spores are useful in production, packaging, delivering and presentation systems for industrial biocatalyst and in medical applications including immunization and vaccination. The spores are also useful as therapeutics and/or prophylactic agents, and as vaccines against a broad spectrum of immunogens and bacterial, viral and parasitic pathogens and toxins. The spores are also useful for production and immobilization of enzymes and proteins for industrial use, and in a variety of biotechnology settings as carriers for nucleic acids and biotin linked ligands. (II) is useful as sensor and detector. (II) is useful as a vaccine or immunomodulatory agent against a disease or disease causing pathogen including Staphylococcus sp., Streptococcus sp., viral encephalitis, human immunodeficiency virus (HIV), cytomegalovirus, poliomyelitus, rabies, cancer, typhoid, parasites, anthrax, foot and mouth disease, Alzheimer's disease, hepatitis, diphtheria, pertussis, hemorrhagic fevers, influenza, cholera, meningitis, measles, mumps, Lyme disease, tetanus, yellow fever and pneumonia. (I) is also useful for treating allergy, asthma, autoimmune diseases, e.g. rheumatoid **arthritis**, diabetes mellitus and multiple sclerosis, septic shock, organ transplantation and inflammatory conditions including inflammatory bowel syndrome, psoriasis, pancreatitis, and other immunodeficiencies.

Dwg.0/12

L9 ANSWER 3 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2

AN 2002:291341 BIOSIS
DN PREV200200291341
TI Expression and regulation of the PD-L1 immunoinhibitory molecule on microvascular endothelial cells.
AU Eppihimer, Michael J. (1); Gunn, Jason; Freeman, Gordon J.; Greenfield, Edward A.; Chernova, Tetyana; Erickson, Jamie; **Leonard, John P.**
CS (1) Discovery Research: Respiratory Diseases, Wyeth/Genetics Institute, Inc., One Burtt Road, Andover, MA, 01810: meppihimer@genetics.com USA
SO Microcirculation (New York), (April, 2002) Vol. 9, No. 2, pp. 133-145. http://www.naturesj.com/mn/index.html. print.
ISSN: 1073-9688.
DT Article
LA English
AB Objective: To evaluate the expression and regulation of a novel B7-like protein, PD-L1, the ligand for the immunoinhibitory receptor PD-1 expressed on activated T-cells, on microvascular endothelial cells (ECs). Methods: PD-L1 expression on ECs in vitro and in vivo was quantified by using a dual radiolabeled **antibody** technique after treatment with interferons (IFN) and **IL-12**, respectively. Changes in the level of PD-L1 mRNA were determined by using RT-PCR. Results: PD-L1 was observed to be present on ECs under basal conditions. Treatment of ECs with IFN-alpha, -beta and -gamma, but not LPS, was observed to induce elevations in the mRNA and surface expression of PD-L1 on ECs. By using a dual radiolabeled monoclonal **antibody** (mAb) technique, PD-L1 expression in various tissues of control and **IL-12** challenged wild-type and IFN-gamma-deficient mice was measured. A significant increase in PD-L1 expression was observed in tissues at 24 hours after **IL-12**-challenge, with peak levels of PD-L1 occurring 72 hours after **IL-12** challenge. **IL-12** was not effective at inducing PD-L1 expression in tissues of IFN-gamma-deficient mice. Conclusions: These data show the expression of a novel B7-like molecule on murine ECs that is mediated by IFN-alpha, -beta, and -gamma, and suggest a potential pathway by which ECs may modulate T-cell function.

L9 ANSWER 4 OF 20 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 2001274332 EMBASE
TI Interleukin-12 gene therapy vaccines: Directing the immune system against minimal residual leukemia.
AU Dunussi-Joannopoulos K.; **Leonard J.P.**
CS Dr. K. Dunussi-Joannopoulos, Genetics Institute, One Burtt Road, Andover, MA 01810, United States
SO Leukemia and Lymphoma, (2001) 41/5-6 (483-492).
Refs: 60
ISSN: 1042-8194 CODEN: LELYEA
CY United Kingdom
DT Journal; General Review
FS 016 Cancer
022 Human Genetics
025 Hematology
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB Current overall survival rates for patients with AML remain poor and there is need for novel therapeutic approaches. One such approach is to use the patient's own immune system to eliminate minimal residual disease. Recent advances in genetic manipulation of tumor cells, together with a better understanding of the immune mechanisms controlling the host-tumor relationship have led to a flurry of preclinical and clinical studies on tumor cell vaccines. Here we present a brief overview of genetic

manipulation of tumor cells, and highlight important principles of cancer immunity and cancer vaccines. Special emphasis is given on recent work on the role of interleukin-12 (**IL-12**) based vaccines in murine AML. These studies have shown that vaccines with AML cells, genetically modified to secrete **IL-12**, are potent stimulators of the immune system and lead to the development of both prophylactic and therapeutic anti-leukemia immunity.

L9 ANSWER 5 OF 20 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 3
AN 2000-524532 [47] WPIDS
DNN N2000-387705 DNC C2000-155840
TI Humanized immunoglobulin having a binding specificity to B7-1 (derived from ATCC PTA-263), or B7-2 (derived from ATCC CRL-12524) molecules, modulates immune responses and can therefore treat e.g. autoimmune diseases, infectious diseases.
DC B04 D16 S03
IN CARRENO, B; CELNIKER, A C; CO, M S; COLLINS, M; FRIEDRICH, S; **GOLDMAN**, **S**; GRAY, G S; KNIGHT, A; OHARA, D; RUP, B; VASQUEZ, M; VELDMAN, G M; WARNER, G; GARVIN, W; GRAY, C S; STUART, F; O'HARA, D
PA (GEMY) GENETICS INST INC
CYC 91
PI WO 2000047625 A2 20000817 (200047)* EN 158p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
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AU 2000039988 A 20000829 (200062)
NO 2001003911 A 20011010 (200174)
EP 1159300 A2 20011205 (200203) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
CZ 2001002925 A3 20020116 (200215)
BR 2000008209 A 20020219 (200222)
ADT WO 2000047625 A2 WO 2000-US3303 20000209; AU 2000039988 A AU 2000-39988 20000209; NO 2001003911 A WO 2000-US3303 20000209, NO 2001-3911 20010810; EP 1159300 A2 EP 2000-919275 20000209, WO 2000-US3303 20000209; CZ 2001002925 A3 WO 2000-US3303 20000209, CZ 2001-2925 20000209; BR 2000008209 A BR 2000-8209 20000209, WO 2000-US3303 20000209
FDT AU 2000039988 A Based on WO 200047625; EP 1159300 A2 Based on WO 200047625; CZ 2001002925 A3 Based on WO 200047625; BR 2000008209 A Based on WO 200047625
PRAI US 1999-339596 19990624; US 1999-249011 19990212
AB WO 200047625 A UPAB: 20000925
NOVELTY - Humanized immunoglobulin having a binding specificity to B7-1 (derived from ATCC PTA-263), or B7-2 (derived from ATCC CRL-12524) molecules, comprising an antigen binding region of non-human origin and a portion of a human immunoglobulin, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a host cell comprising nucleic acid that encodes a humanized B7-1 **antibody** and/or a humanized B7-2 **antibody**;

(2) a humanized immunoglobulin light/heavy chain having binding specificity for B7-1 comprising CDR1, CDR2, and CDR3 of the light/heavy chain of murine 1F1 **antibody** and a human light/heavy chain framework region;

(3) an isolated nucleic acid (N1) comprising a defined 390 base pair (bp) sequence, encoding a defined 130 amino acid human immunoglobulin light chain variable region (P1) of B7-1 (both given in the specification);

(4) an isolated nucleic acid (N2) comprising a defined 405 base pair

(bp) sequence, encoding a defined 135 amino acid human immunoglobulin heavy chain variable region (P2) of B7-1 (both given in the specification);

(5) an isolated nucleic acid (N3) comprising a defined 396 base pair (bp) sequence, encoding a defined 132 amino acid human immunoglobulin light chain variable region (P3) of B7-2 (both given in the specification);

(6) an isolated nucleic acid (N4) comprising a defined 405 base pair (bp) sequence, encoding a defined 135 amino acid human immunoglobulin heavy chain variable region (P4) of B7-2 (both given in the specification);

(7) a fused gene encoding humanized immunoglobulin light or heavy chain comprising a first nucleic acid sequence encoding an antigen binding region derived from murine 1F1 or 3D1 monoclonal **antibody** and a second nucleic acid sequence encoding a portion of a constant region of an immunoglobulin of human origin;

(8) a method for inhibiting the interaction of a first cell bearing a B7-1 receptor with a second cell bearing B7-1, comprising contacting the first cell with a humanized immunoglobulin having a binding specificity to B7-1, or B7-2 molecules;

(9) a method for treating an individual having a transplanted organ, tissue or cell comprising administering humanized immunoglobulin having a binding specificity to B7-1, or B7-2 molecules;

(10) a method for treating a disease modulated by B7-1 or B7-2;

(11) a method for making a humanized immunoglobulin having binding specificity for B7-1 or B7-2 comprising:

(a) determining the complementarity determining regions (CDRs) of an **antibody** of non-human origin which has binding specificity for B7-1 or B7-2;

(b) obtaining a human **antibody** having a framework region amino acid sequence suitable for grafting of the CDRs in (a); and

(c) grafting the CDRs of (a) with those of (b);

(12) a method for determining the presence or absence of B7-1 or B7-2 in a sample comprising:

(a) contacting the sample with an **antibody** specific to B7-1 or B7-2 to allow complex formation; and

(b) detecting the presence or absence of the complex;

(13) a humanized immunoglobulin light or heavy chain having binding specificity for B7-2 comprising CDR1, CDR2, and CDR3 of the light chain of murine 3D1 **antibody**, and a human light or heavy chain framework region;

(14) a method for transplanting cells into an individual comprising:

(a) obtaining cells from a donor;

(b) contacting the cells with an immunoglobulin specific to B7-1 and B7-2 and recipient cells from the individual to allow tolerance reduction; and

(c) introducing the mixture to the individual;

(15) a method for treating a disorder selected from autoimmune diseases, infectious diseases, inflammatory disorders, systemic lupus erythematosus, diabetes mellitus, insulitis, asthma, **arthritis**, inflammatory bowel disease, inflammatory dermatitis, and multiple sclerosis comprising administering a humanized immunoglobulin to B7-1 and B7-2

(16) a method for treating a transplant recipient or preventing transplant rejection in a transplant recipient, comprising administering an immunoglobulin specific to B7-1 and B7-2; and

(17) a method for decreasing an **antibody** response to an antigen in a mammal comprising administering a humanized immunoglobulin specific to B7-1 or B7-2.

ACTIVITY - Immunosuppressive; antiinfective; antiinflammatory; dermatological; antidiabetic; antiasthmatic; antiarthritic; cytostatic; antianemic; neuroprotective.

MECHANISM OF ACTION - Modulation of immune responses; inhibition of T cell costimulation.

Isolated CD28+ T cells were washed once and resuspended in RPMI (not defined) complete medium, supplemented with 2 ng/ml PMA (not defined), to a cell density of 5 multiply 105 cells/ml. The CD28+ T cells were added to the antibody/CHO/hB7-2 mixture, incubated for 3 days at 37 deg. C, 5% CO2, and T cell proliferation was measured by pulsing for the last 6 hours of culture with 1 uCi of (3H)-thymidine. The cells were harvested on a filter and the incorporated radioactivity was measured in a scintillation counter. Results showed that both antibodies exhibited dose dependent inhibition of B7-2 driven T cell proliferation with similar IC50 (inhibitory concentration 50%) values of 72 pm (murine anti-hB7-2) and 50 pm (humanized anti-hB7-2) indicating that both antibodies were similar and very effective in inhibiting the B7-2 T cell stimulatory signal. This demonstrated that the high affinity anti-B7-2 mAbs could block B7-2 functionality by inhibiting the activation and/or proliferation of human T cells.

USE - The humanized immunoglobulin with binding specificity to B7-1 and/or B7-2 is useful for treating autoimmune diseases, infectious diseases, inflammatory disorders, systemic lupus erythematosus, diabetes mellitus, insulitis, asthma, arthritis, inflammatory bowel disease, inflammatory dermatitis, and multiple sclerosis. The immunoglobulins are also useful for treating a transplant recipient or preventing transplant rejection in a transplant recipient, and treating proliferative disease (leukemia, lymphoma and cancer), anemia (sickle-cell anemia, thalassemia and aplastic anemia), inborn errors of metabolism, congenital immunodeficiency diseases, and myeloid dysplasia syndrome.

Dwg.0/28

L9 ANSWER 6 OF 20 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 4
AN 2000-430395 [37] WPIDS
CR 1997-132638 [12]; 1997-165283 [15]; 2000-282570 [23]
DNC C2000-130765
TI New polynucleotides encoding human cytotoxic T-lymphocyte antigen (CTLA)-8 proteins useful for treating e.g. autoimmune diseases, inflammatory diseases, and microbial infections.
DC B04 D16
IN CARLIN, M; GIANNOTTI, J; GOLDEN-FLEET, M M; **GOLDMAN**, **S**; JACOBS, K; KELLEHER, K; MI, S; NEBEN, S; PITTMAN, D
PA (GEMY) GENETICS INST INC
CYC 1
PI US 6074849 A 20000613 (200037)* 25p
ADT US 6074849 A Provisional US 1995-35347P 19950719, CIP of US 1995-514014 19950811, US 1996-685239 19960718
PRAI US 1995-35347P 19950719; US 1995-514014 19950811; US 1996-685239 19960718
AB US 6074849 A UPAB: 20010711

NOVELTY - An isolated polynucleotide (I) comprising nucleotides 146-544 of an 813 base pair sequence, fully defined in the specification, or its variant resulting from degeneracy of the genetic code, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a host cell transformed with (I) operably linked to an expression control sequence; and

(2) producing a human cytotoxic T-lymphocyte antigen (CTLA)-8 protein comprising culturing a host cell of (1), and purifying the human CTLA-8 protein from the culture.

ACTIVITY - Immunosuppressive; neuroprotective; dermatological; antirheumatic; antiarthritic; antithyroid; antidiabetic; ophthalmological; anti-HIV; antibacterial; hepatotropic; anti-inflammatory; virucide; antifungal; protozoacide; antiallergic; antianemic; cytostatic.

MECHANISM OF ACTION - Gene therapy; Interferon production inducer;

Interleukin (IL)-3 inducer; granulocyte-monocyte colony stimulating factor inducer; chemotaxis-stimulator. MRC5 cells were incubated in the presence of human CTLA-8 (B18), and production of IL-6 and IL-8 were measured. Herpes CTLA-8 (IL-7) was used as positive control. B18 showed titerable production of both IL-6 and IL-8.

USE - (I) is useful for treating autoimmune disorders (e.g. multiple sclerosis, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune thyroiditis, insulin dependent diabetes mellitus, myasthenia gravis, and autoimmune inflammatory eye disease), viral (e.g. human immunodeficiency virus (HIV), hepatitis, herpes, influenza, cytomegalovirus (CMV)), bacterial, fungal (e.g. Candida) or protozoan (e.g. malaria, leshmaniasis) infections. (I) can also be used for treating allergic reactions, and to suppress chronic or acute inflammation associated with infection such as septic shock or systemic inflammatory response syndrome, inflammatory bowel disease, Crohn's disease or resulting from over production cytokines, regulation of hematopoiesis and consequently in the treatment of myeloid or lymphoid cell deficiencies, supports the growth and proliferation of erythroid progenitor cells alone or in combination with other cytokines. (I) can be used in treating anemia, in chemotherapy to treat or prevent consequent myeloid suppression, in treating various stem cell disorders (e.g. aplastic anemia and paroxysmal nocturnal hemoglobinuria), and in repopulating the stem cell compartment post irradiation/chemotherapy, either in vivo or ex vivo as normal cells or genetically manipulated for gene therapy. Human CTLA-8 proteins may be used to inhibit growth and proliferation of vascular endothelial cells, hence effective in inhibiting angiogenesis as well as in treating tumors, as well as to immunize animals to obtain **antibodies** which specifically react with the CTLA-8 protein, and which may inhibit CTLA-8 binding to its receptor.

Dwg.0/10

L9 ANSWER 7 OF 20 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 5
AN 2000-282570 [24] WPIDS
CR 1997-132638 [12]; 1997-165283 [15]; 2000-430395 [36]
DNC C2000-085202
TI Novel human CTLA-8 protein useful for treating immunodeficiencies and disorders, in regulating growth, proliferation and/or activity of T and/or B lymphocytes and multiple sclerosis, rheumatoid **arthritis**.
DC B04 D16
IN CARLIN, M; GIANNOTTI, J; GOLDEN-FLEET, M M; **GOLDMAN**, **S**; JACOBS, K; KELLEHER, K; MI, S; NEBEN, S; PITTMAN, D
PA (GEMY) GENETICS INST INC
CYC 1
PI US 6043344 A 20000328 (200024)* 25p
ADT US 6043344 A Provisional US 1995-35347P 19950719, CIP of US 1995-504032 19950719, CIP of US 1995-514014 19950811, Div ex US 1996-685239 19960718, US 1998-34810 19980304
FDT US 6043344 A CIP of US 5707829
PRAI US 1995-35347P 19950719; US 1995-504032 19950719; US 1995-514014 19950811; US 1996-685239 19960718; US 1998-34810 19980304
AB US 6043344 A UPAB: 20010711

NOVELTY - An isolated human CTLA-8 (B18) (I) with a fully defined sequence as given in the specification, is new.

DETAILED DESCRIPTION - (I) comprises a fully defined sequence of 163 (2) amino acids, amino acids 11-, 29- or 31-163 of (2), the fragment of (2) comprising amino acids 11-, 29- or 31-163 of (2), as given in the specification.

An INDEPENDENT CLAIM is also included for the preparation of (I).

ACTIVITY - Immunosuppressive; antiarthritic; antiinflammatory; immunostimulant; antidiabetic; neuroprotective; dermatological; antianemic; antiallergic; antithyroid; antiasthmatic; antibacterial; cytostatic.

MECHANISM OF ACTION - Angiogenesis inhibitor; hematopoiesis regulator; growth or proliferation of vascular endothelial cells inhibitor; tumor growth inhibitor; myeloid, lymphoid cells or their progenitors proliferator; IFN- gamma , IL-3, GM-CSF production inducer; gene therapy. The ability of (I) to inhibit angiogenesis was examined in an angiostatic activity assay. Primary human umbilical cells (HUVECs) were seeded to 2 multiply 103 cells/well of a 96 well plate and incubated. The cells were then starved in M199 medium. Conditioned media containing B18 was obtained from transfected COS or stably expressing CHO cells and 1:10, 1:50, 1:250 and 1:1250 were prepared in M199-CS medium containing 100 ng/ml FGF. The dilutions of B18 were added to the starved cells and incubated for 72 hr at 37 deg. C. The cells were then radiolabeled and trypsinized for liquid scintillation counting, after washing. Results showed that human CTLA-8 (B18) inhibits angiogenesis.

USE - (I) is used for treating immune deficiencies and disorders (including severe combined immunodeficiency (SCID), e.g. in regulating growth and proliferation of T and/or B lymphocytes, and effecting the cytolytic activity of NK cells and other cell populations. These immune deficiencies may be genetic or caused by viruses, bacterial or fungal infections. The proteins are also used for boosting the immune system for treating cancer and in the treatment of autoimmune diseases such as multiple sclerosis, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent diabetes mellitis, myasthenia gravis, graft-versus-host disease and autoimmune inflammatory eye disease. They are also used for treating asthma, allergic reactions or other respiratory problems and suppressing chronic or acute inflammation associated with infection such as septic shock or systemic inflammatory response syndrome (SIRS), inflammatory bowel disease and Crohn's disease. (I) is also used for regulating hematopoiesis and consequently in the treatment of myeloid or lymphoid deficiencies i.e. by supporting the growth and proliferation of erythroid progenitor cells, myeloid cells, megakaryocytes, hematopoietic stem cells and thus used for treating anemia, thrombocytopenia, aplastic anemia and paroxysmal nocturnal hemoglobinuria. They also inhibit the growth and proliferation of vascular endothelial cells and thus are effective in inhibiting angiogenesis. The polynucleotides encoding (I) can be used in gene therapy. The proteins are used as immunogens to produce polyclonal or monoclonal **antibodies** useful for performing diagnostic assays for CTLA-8.

DESCRIPTION OF DRAWING(S) - The figure shows the data relating to the ability of CTLA-8 to inhibit angiogenesis.

Dwg.3/7

L9 ANSWER 8 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2001:39565 BIOSIS
DN PREV200100039565
TI Myelin oligodendrocyte glycoprotein induced EAE in **IL-12** p35 deficient mice.
AU Hunter, S. E. (1); Thibodeaux, D. K. (1); Bouchard, P. (1); **Leonard, J. P. (1)**
CS (1) Genetics Institute, Inc., Cambridge, MA, 02140 USA
SO FASEB Journal, (April 20, 2000) Vol. 14, No. 6, pp. A1116. print.
Meeting Info.: Joint Annual Meeting of the American Association of Immunologists and the Clinical Immunology Society Seattle, Washington, USA May 12-16, 2000
ISSN: 0892-6638.
DT Conference
LA English
SL English

L9 ANSWER 9 OF 20 MEDLINE
AN 2000021818 MEDLINE

TI Immunological reconstitution and correlation of circulating serum inflammatory mediators/cytokines with the incidence of acute graft-versus-host disease during the first 100 days following unrelated umbilical cord blood transplantation.

AU Abu-Ghosh A.; **Goldman S.**; Slone V.; Van de Ven C.; Suen Y.; Murphy L.; Sender L.; Cairo M.S.

CS Dr. M.S. Cairo, Georgetown University Medical Center, Lombardi Cancer Center, 2 East Main, 3800 Reservoir Rd. NW, Washingon DC 20007, United States

SO Bone Marrow Transplantation, (1999) 24/5 (535-544).
Refs: 39
ISSN: 0268-3369 CODEN: BMTRE

CY United Kingdom

DT Journal; Article

FS 005 General Pathology and Pathological Anatomy
016 Cancer
025 Hematology
026 Immunology, Serology and Transplantation
037 Drug Literature Index

LA English

SL English

AB We investigated early immunological reconstitution and the production of circulating inflammatory mediators and their relationship to aGVHD in children during the first 100 days following unrelated UCBT. Nine patients had an underlying malignant disease (ALL, ANLL), and two, non-malignant diseases (SAA, ALD). The median age was 10 years (range: 1.25-21). Seven of 11 patients were alive by day 100, two died from regimen-related toxicity, and two died from severe aGVHD (grade .gtoreq. III). Myeloid engraftment (ANC .gtoreq. 500 /mm3 x 2 days) occurred at a median of 24 days (range: 14-55), while platelet engraftment (platelet count .gtoreq. 20,000 /mm3 untransfused x 7 days) was delayed and occurred at a median of 52 days (range: 33-95). The mean cell dose of CD34+ cells was 3.3 .+-. 3.51 x 105 /kg, and of CD34+/CD41+ cells was 3.94 .+-. 3.99 x 104 /kg. Acute GVHD (grade II-IV) developed in seven patients (77%), and severe aGVHD (grade III-IV) developed in five patients (55%). Serum levels of IL-2R.alpha.; IL-2, IL-4, IL-7, **IL-12**, and IFN.gamma.
were not significantly different between patients with grades 0-I aGVHD and patients with grades II-IV aGVHD. Evaluation of immunological reconstitution on day 90 post UCBT demonstrated an early recovery of the absolute numbers of B cells (CD19+) and NK cells (CD3-/CD56+). Immunoglobulin levels for IgG, IgM and IgA remained normal throughout the study period. PMN functional studies demonstrated normal superoxide generation, bacterial killing (BK), and chemotaxis (CTX). However, both helper (CD3+/CD4+) and suppressor (CD3+/CD8+)T cell subsets remained low during the first 100 days post UCBT with mean .+-. s.e.m. values of 120 .+-. 29 /mm3 and 10 .+-. 50 /mm3, respectively (normal = 900-2860 /mm3 (CD3/CD4), normal = 630-1910 /mm3 (CD3/CD8)). Mitogen response studies showed low blastogenesis to PHA and PWM, with a mean c.p.m. .+-. s.e.m. value of 1.7 .+-. 0.67 x 104 for PHA (NL .gtoreq. 75 x 103) and 8.42 .+-. 4.1 x 103 for PWM (NL .gtoreq. 25 x 103). In conclusion, serum levels of inflammatory mediators were not predictive nor did they correlate with the severity of aGVHD. Recovery of NK cells, B cells, and PMN functions occurred within the first 90 days post transplant. However, T cell subsets, CD3+/CD4+ and CD3+/CD8+, and T cell functional activity remained significantly decreased and may account for the high incidence of infectious morbidity seen during this immediate post UCBT period.

L9 ANSWER 12 OF 20 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 6

AN 1997-132638 [12] WPIDS

CR 1997-165283 [15]; 2000-282570 [23]; 2000-430395 [36]

DNC C1997-042879

TI New nucleic acid encoding the CTLA-8 protein - modulates growth of

DN 20021818 PubMed ID: 10553047
TI Autocrine regulation of **IL-12** receptor expression is independent of secondary IFN-gamma secretion and not restricted to T and NK cells.
AU Thibodeaux D K; Hunter S E; Waldburger K E; Bliss J L; Trepicchio W L; Sypek J P; Dunussi-Joannopoulos K; Goldman S J; **Leonard J P**
CS Preclinical Research and Development, Genetics Institute, Andover, MA 01810, USA.
SO JOURNAL OF IMMUNOLOGY, (1999 Nov 15) 163 (10) 5257-64.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199912
ED Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991202
AB The biological response to **IL-12** is mediated through specific binding to a high affinity receptor complex composed of at least two subunits (designated IL-12Rbeta1 and IL-12Rbeta2) that are expressed on NK cells and activated T cells. The selective loss of IL-12Rbeta2 expression during Th2 T cell differentiation suggests that regulation of this receptor component may govern **IL-12** responsiveness. In murine assays, down-regulation of IL-12Rbeta2 expression can be prevented by treatment with IFN-gamma, indicating that receptor expression and hence **IL-12** responsiveness may be regulated, at least in part, by the local cytokine milieu. In this study, we report that cellular expression of both IL-12Rbeta1 and beta2 mRNA is increased in the lymph nodes of naive mice following systemic administration of murine rIL-12 (rmIL-12). Changes in IL-12R mRNA were associated with increased IFN-gamma secretion following ex vivo activation of lymph node cells with rmIL-12, indicating the presence of a functional receptor complex. Expression of IL-12R mRNA was not restricted to lymph node T cells, and its autocrine regulation was independent of secondary IFN-gamma secretion. Data from fractionated lymph node cells as well as rmIL-12-treated B cell-deficient mice suggest that **IL-12** -responsive B cells may represent an alternative cellular source for IFN-gamma production. However, the strength of the biological response to rmIL-12 is not governed solely by receptor expression, as rmIL-12-induced IFN-gamma secretion from cultured lymph node cells is accessory cell dependent and can be partially blocked by inhibition of B7 costimulation.

L9 ANSWER 10 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:275655 BIOSIS
DN PREV199900275655
TI Prolonged inhibition of murine lupus by short term therapy with anti-B7 and anti-**IL-12 antibodies** during onset of disease.
AU Collins, M. (1); Nagle, S. (1); Chung, C. (1); **Goldman, S. (1);** Sypek, J. (1)
CS (1) Genetics Institute, Andover, MA, 01810 USA
SO FASEB Journal, (March 15, 1999) Vol. 13, No. 5 PART 2, pp. A956.
Meeting Info.: Annual Meeting of the Professional Research Scientists on Experimental Biology 99 Washington, D.C., USA April 17-21, 1999 Federation of American Societies for Experimental Biology
. ISSN: 0892-6638.
DT Conference
LA English

L9 ANSWER 11 OF 20 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 1999315480 EMBASE

vascular endothelial and haematopoietic cells and induces cytokine expression, for treating infection, auto-immune disease, etc..

DC B04 D16

IN CARLIN, M; JACOBS, K; KELLEHER, K; MCCOY, J M; GIANNOTTI, J; GOLDEN-FLEET, M; **GOLDMAN, S**; MI, S; NEBEN, S; PITTMAN, D; DUCKETT, M C; GOLDEN-FLEET, M M; PITMAN, D; CARLIN-DUCKETT, M

PA (GEMY) GENETICS INST INC

CYC 23

PI WO 9704097 A2 19970206 (199712)* EN 50p
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AU CA JP MX
AU 9667123 A 19970218 (199723)
WO 9704097 A3 19970912 (199749)
US 5707829 A 19980113 (199809) 30p
EP 839196 A2 19980506 (199822) EN
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
JP 11510045 W 19990907 (199947) 59p
US 5969093 A 19991019 (199950)
MX 9800507 A1 19980501 (200007)
MX 9801120 A1 19990401 (200055)
AU 727480 B 20001214 (200103)
AU 727489 B 20001214 (200103)
AU 2001028001 A 20010517 (200138)#
AU 2001028002 A 20010802 (200152)#

ADT WO 9704097 A2 WO 1996-US11889 19960718; AU 9667123 A AU 1996-67123 19960218; US 5707829 A US 1995-514014 19950811; EP 839196 A2 EP 1996-927237 19960718, WO 1996-US11889 19960718; JP 11510045 W WO 1996-US11889 19960718, JP 1997-506846 19960718; US 5969093 A Div ex US 1995-514014 19950811, US 1997-833823 19970410; MX 9800507 A1 MX 1998-507 19980116; MX 9801120 A1 MX 1998-1120 19980210; AU 727480 B AU 1996-67123 19960718; AU 727489 B AU 1996-67685 19960808; AU 2001028001 A Div ex AU 1996-67685 19960808, AU 2001-28001 20010314; AU 2001028002 A Div ex AU 1996-67123 19960718, AU 2001-28002 20010314

FDT AU 9667123 A Based on WO 9704097; EP 839196 A2 Based on WO 9704097; JP 11510045 W Based on WO 9704097; AU 727480 B Previous Publ. AU 9667123, Based on WO 9704097; AU 727489 B Previous Publ. AU 9667685, Based on WO 9707198; AU 2001028001 A Div ex AU 727489; AU 2001028002 A Div ex AU 727480

PRAI US 1995-514014 19950811; US 1995-504032 19950719; US 1997-833823 19970410; WO 1996-US12897 19960808; AU 2001-28001 20010314; AU 2001-28002 20010314

AB WO 9704097 A UPAB: 20011001

A novel isolated polynucleotide (I) comprises: (a) nucleotides (nt) 146-544 of an 813 bp sequence given in the specification; (b) a sequence able to hybridise with (a) or varying from (a) only within the degeneracy of the genetic code; or (c) an allelic variant of (a). Also claimed are: (1) host cells transformed with (I); (2) isolated human CTLA-8 protein which has 163 amino acids (aa), its 11-163, 29-163 or 31-163 regions or any fragments of them with CTLA-8 activity; and (3) **antibodies** (Ab) which specifically react with CTLA-8 protein.

USE - (I) encodes proteins with CTLA-8 activity. Treatment of mammals with CTLA-8 (or non-human analogues or IL-17) results in at least one of: (a) inhibition of angiogenesis, growth/proliferation of vascular endothelial cells, tumour cells and angiogenesis-dependent tissue growth; (b) proliferation of myeloid, erythroid or lymphoid cells (or their progeny); or (c) induction of interferon- gamma , IL-3 or GM-CSF prodn (claimed). Opt. CTLA-8 is expressed in vivo from a suitable vector. Typical applications of CTLA-8 are treatment of immune deficiency and disorders requiring modulation of T/B cell growth or proliferation, or of cytolytic natural killer cells, e.g. viral or microbial infection (e.g. HIV, hepatitis, malaria, candidiasis etc.); autoimmune disease (e.g. multiple sclerosis, rheumatoid **arthritis**, insulin-dependent

diabetes etc.); to boost the immune response in cancer treatment; as antiinflammatories (e.g. in septic shock or Crohn's disease) and in haematopoietic disorders where growth/proliferation of erythroid, myeloid or megakaryocytic cells is needed. Ab can be used to determine CTLA-8, possibly also for treating some tumours or some of the above conditions. Dwg.0/7

L9 ANSWER 13 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 7
AN 1998:80243 BIOSIS
DN PREV199800080243
TI Immunoregulation by interleukin-12 in MB49.1 tumor bearing mice: Cellular and cytokine-mediated effector mechanisms.
AU Hunter, Sharon E.; Waldburger, Kristine E.; Thibodeaux, Deborah K.; Schaub, Robert G.; **Goldman, Samuel J.**; **Leonard, John P.** **(1)**
CS (1) Genet. Inst., One Burtt Rd., Andover, MA 01810 USA
SO European Journal of Immunology, (Dec., 1997) Vol. 27, No. 12, pp. 3438-3446.
ISSN: 0014-2980.
DT Article
LA English
AB Administration of recombinant murine interleukin (rmIL)-12 to MB49.1 tumor-bearing mice results in dose-dependent regression of the primary tumor and the generation of protective antitumor immunity in the majority of animals. rmIL-12 administration is associated with a marked increase in lymph node cellularity that is predominantly due to the expansion of B220+ B cells as well as CD8+ T cells. Stimulation of lymph node cells from rmIL-12-treated, but not control tumor-bearing mice, with MB49.1 tumor cells in vitro was shown to enhance the secretion of interferon (IFN)-gamma. The magnitude of this in vitro response was dependent on the dose of rmIL-1 2 administered in vivo and mirrored the change in circulating serum IFN-gamma. Furthermore, at the height of the in vitro response to tumor stimulation, the addition of a neutralizing **antibody** to murine **IL-12** suppressed IFN-gamma production, indicating a role for endogenous **IL-12** in this antigen-specific cytokine response. Although studies in SCID mice confirmed that an appropriate T cell response was required for rmIL-12-mediated antitumor activity, in immunocompetent animals early tumor regression was not accompanied by cellular infiltration of the tumor. In contrast, a profound increase in tumor-associated inducible nitric oxide synthase (iNOS) was observed in mice receiving rmIL-12 which preceded T cell infiltration of the tumor which could be detected during the second week of **IL-12** treatment. Direct tumor killing through the cytotoxic actions of NO via the iNOS pathway may serve as a way of generating tumor antigen which enables the host to mount a subsequent T cell response against the tumor.

L9 ANSWER 14 OF 20 MEDLINE DUPLICATE 8
AN 1998080712 MEDLINE
DN 98080712 PubMed ID: 9419442
TI Regulation of the inflammatory response in animal models of multiple sclerosis by interleukin-12.
AU **Leonard J P**; Waldburger K E; Schaub R G; Smith T; Hewson A K; Cuzner M L; Goldman S J
CS Genetics Institute, Andover, MA 01810, USA.
SO CRITICAL REVIEWS IN IMMUNOLOGY, (1997) 17 (5-6) 545-53. Ref: 54
Journal code: 8914819. ISSN: 1040-8401.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

LA English
FS Priority Journals
EM 199802
ED Entered STN: 19980306
Last Updated on STN: 20000303
Entered Medline: 19980225
AB Interleukin 12 (**IL-12**), a novel heterodimeric protein produced primarily by antigen-presenting cells, serves as a key regulator of innate and adaptive immune responses. In addition to being a potent inducer of IFN-gamma, **IL-12** is widely considered to be the principal cytokine that regulates the generation of Th1 type effector cells. As the successful induction of experimental autoimmune encephalomyelitis (EAE) is associated with a strong Th1 type cellular response, we have evaluated the role of **IL-12** in regulating the pathogenesis of EAE in SJL/J mice and Lewis rats. In both settings, treatment with **IL-12** was found to accelerate the onset and increase the severity and duration of clinical disease. More importantly, administration of **IL-12** to Lewis rats that had recovered from primary disease was found to trigger clinical relapse. In all instances, **IL-12**-induced exacerbation was associated with a profound increase in iNOS positive macrophages within the perivascular lesions. Although **IL-12** -induced IFN-gamma does not appear to be required for exacerbation of disease, neutralizing **antibodies** against murine **IL-12** delay the onset and reduce the severity of adoptively transferred EAE, indicating a role for endogenous **IL-12** as regulator of disease. Based on the above findings, effective inhibition of **IL-12** in vivo may have great therapeutic value in the treatment of MS and other Th1-associated inflammatory disorders.

L9 ANSWER 15 OF 20 MEDLINE DUPLICATE 9
AN 97118050 MEDLINE
DN 97118050 PubMed ID: 8958933
TI Regulation of experimental autoimmune encephalomyelitis by interleukin-12.
AU **Leonard J P**; Waldburger K E; Goldman S J
CS Genetics Institute, Andover, Massachusetts 01810, USA.
SO ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (1996 Oct 31) 795 216-26.
Journal code: 7506858. ISSN: 0077-8923.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; AIDS
EM 199701
ED Entered STN: 19970128
Last Updated on STN: 20000303
Entered Medline: 19970108
AB We have evaluated the effects of rmIL-12 on the course of adoptively transferred EAE. When mice were injected with LNC that had been stimulated in vitro with PLP in the presence of rmIL-12, the subsequent course of disease was more severe and prolonged than controls. In vitro stimulation with PLP in the presence of **IL-12** was associated with an increase in IFN-gamma and decrease in IL-4-producing cells, indicating a preferential expansion of Th1 effector cells. At peak disease, no notable differences in either the cellular composition or cytokine expression within CNS lesions was seen between groups. However, the frequency of macrophages that stained positively for inducible nitric oxide synthase (iNOS) was increased in animals challenged with rmIL-12 treated LNC. These data suggest that in addition to promoting the preferential expansion of IFN-gamma-producing cells by rmIL-12 treatment in vitro, in vivo effects leading to macrophage activation and iNOS expression may contribute to the severe, protracted course of CNS inflammation in this model. In contrast, treatment of mice with an

antibody to murine **IL-12** following cell transfer completely prevented paralysis with only 40% of the mice developing mild disease. These data suggest that endogenous **IL-12** plays a pivotal role in the pathogenesis of this model of autoimmune disease.

L9 ANSWER 16 OF 20 CAPLUS COPYRIGHT 2002 ACS
AN 1997:285388 CAPLUS
DN 126:329093
TI Effects of interleukin 12 on hematopoietic stem and progenitor cells
AU Neben, Steven; Leonard, John; **Goldman, Samuel**; Ploemacher, Rob E.
CS Department of Immunology and Hematopoiesis, Genetics Institute, Inc., Cambridge, MA, USA
SO Bone Marrow Transplantation: Basic and Clinical Studies, [Papers presented at the International Symposium on BMT--Basic and Clinical Studies], Tokyo, Oct. 9-10, 1995 (1996), Meeting Date 1995, 28-35. Editor(s): Ikehara, Susumu; Takaku, Fumimaro; Good, Robert A. Publisher: Springer, Tokyo, Japan.
CODEN: 64HVAW
DT Conference; General Review
LA English
AB A review with 34 refs. Interleukin-12 (**IL-12**) has been shown to possess potent immunomodulatory activity. It has a unique structure among cytokines, consisting of two covalently linked subunits, one with homol. to other members of the cytokine superfamily, the other being highly homologous to gp130, the signaling subunit of a no. of cytokine receptors. Here we summarize studies showing that **IL-12** is a hematopoietic growth factor with potent activity on hematopoietic stem and progenitor cells. In clonal and liq. culture assays, **IL-12** synergizes with IL-3 and Steel Factor to increase the no. of colonies as well as to expand both stem and progenitor cell content in the cultures. In stroma-dependent long-term bone marrow cultures, **IL-12** addn. causes a decrease in cell prodn. in the first week after inoculation of whole bone marrow cells, followed by an increase in both mature cells and progenitor cells over the next 3 wk. The initial decrease appears to be mediated by **IL-12**-induced prodn. of IFN-.gamma., possibly by natural killer cells and/or T cells which do not persist in these cultures. Studies in naive mice demonstrate a similar acute decrease in peripheral leukocyte count, mediated by IFN-.gamma., upon administration of **IL-12**. In contrast, despite a significant decrease in peripheral platelet count, reticulated platelets become elevated and mean megakaryocyte ploidy in the bone marrow shifts from 16N to 32N during **IL-12** treatment. These **IL-12**-mediated effects on megakaryopoiesis are abrogated by simultaneous treatment of mice with **antibodies** against IFN-.gamma.. These studies provide further information on the potential physiol. role and applications of **IL-12** outside the immune system.

L9 ANSWER 17 OF 20 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 10
AN 1995-336810 [43] WPIDS
DNC C1995-148498
TI Use of interleukin-I2 or an Il-I2 **antagonist** - for treating autoimmune conditions, eg. multiple sclerosis, lupus, rheumatoid **arthritis** or diabetes.
DC B04
IN **GOLDMAN, S; LEONARD, J P; OHARA, R**; LEONARD, J; O'HARA, R
PA (GEMY) GENETICS INST INC
CYC 24
PI WO 9524918 A1 19950921 (199543)* EN 37p

RW: AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE
W: AU CA JP
AU 9519749 A 19951003 (199602)
ZA 9500960 A 19951227 (199605) 33p
EP 750509 A1 19970102 (199706) EN
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE
JP 09510444 W 19971021 (199801) 33p
AU 689236 B 19980326 (199826)
IL 112677 A 20000131 (200015)
TW 400233 A 20000801 (200109)
US 6338848 B1 20020115 (200208)
EP 1179348 A2 20020213 (200219) EN
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE
EP 750509 B1 20020515 (200234) EN
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE

ADT WO 9524918 A1 WO 1995-US2550 19950307; AU 9519749 A AU 1995-19749 19950307; ZA 9500960 A ZA 1995-960 19950207; EP 750509 A1 EP 1995-912666 19950307, WO 1995-US2550 19950307; JP 09510444 W JP 1995-524044 19950307, WO 1995-US2550 19950307; AU 689236 B AU 1995-19749 19950307; IL 112677 A IL 1995-112677 19950216; TW 400233 A TW 1995-101380 19950214; US 6338848 B1 Cont of US 1994-212629 19940314, Cont of US 1995-560943 19951120, US 2000-513380 20000225; EP 1179348 A2 Div ex EP 1995-912666 19950307, EP 2001-117762 19950307; EP 750509 B1 EP 1995-912666 19950307, WO 1995-US2550 19950307, Related to EP 2001-117762 19950307

FDT AU 9519749 A Based on WO 9524918; EP 750509 A1 Based on WO 9524918; JP 09510444 W Based on WO 9524918; AU 689236 B Previous Publ. AU 9519749, Based on WO 9524918; EP 1179348 A2 Div ex EP 750509; EP 750509 B1 Related to EP 1179348, Based on WO 9524918

PRAI US 1994-212629 19940314; US 1995-560943 19951120; US 2000-513380 20000225

AB WO 9524918 A UPAB: 19951102
A method for treating in a mammalian subject an autoimmune condition comprises administering (i) an interleukin-I2 (IL-I2) **antagonist** or (ii) IL-I2.

USE - The method is used partic. for autoimmune conditions which are promoted by increased levels of TNF-alpha or IFN-gamma (claimed). It can be used for treating multiple sclerosis, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune pulmonary inflammation, Ciuillan-Barre syndrome, autoimmune thyroiditis, insulin dependent diabetes mellitus or autoimmune inflammatory eye disease (claimed). Dwg.0/6

L9 ANSWER 18 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 11

AN 1995:101409 BIOSIS

DN PREV199598115709

TI Prevention of experimental autoimmune encephalomyelitis by **antibodies** against interleukin 12.

AU **Leonard, J. P. (1)**; Waldburger, K. E.; Goldman, S. J.

CS (1) Genetics Inst., Preclinical Biol., 87 Cambridge Park Dr., Cambridge, MA 02140 USA

SO Journal of Experimental Medicine, (1995) Vol. 181, No. 1, pp. 381-386. ISSN: 0022-1007.

DT Article

LA English

AB Experimental allergic encephalomyelitis (EAE) is an autoimmune disease of the central nervous system that can be transferred to naive mice via CD4+ T cells isolated from appropriately immunized mice. We have evaluated the effects of recombinant murine interleukin 12 (rmIL-12), a potent inducer of interferon gamma (IFN-gamma) and promoter of Th1 T cell development, on the course of adoptively transferred EAE. The transfer of lymph node cells (LNC) isolated from proteolipid protein (PLP)-primed animals and

stimulated in vitro with PLP to naive mice resulted in a progressive paralytic disease culminating in complete hind limb paralysis in the majority of the recipients. When mice were injected with LNC that had been stimulated in vitro with PLP in the presence of rmIL-12, the subsequent course of disease was more severe and prolonged. The addition of rmIL-12 during the in vitro stimulation with PLP resulted in a 10-fold increase in IFN-gamma and a 2-fold increase in tumor necrosis factor (TNF) a in the supernatants, relative to LNC stimulated with PLP alone. However, neutralization of IFN-gamma or TNF-alpha in vitro with specific **antibodies** did not abrogate the ability of rmIL-12 to exacerbate the subsequent disease. Similarly, mice treated with rmIL-12 in vivo after the transfer of antigen-stimulated LNC developed a more severe and prolonged course of disease compared with vehicle-treated control animals. In contrast, treatment of mice with an **antibody** to murine **IL-12** after cell transfer completely prevented paralysis, with only 40% of the mice developing mild disease. These results demonstrate that in vitro stimulation of antigen primed LNC with PLP and rmIL-12 enhances their subsequent encephalitogenicity. Furthermore, inhibition of endogenous **IL-12** in vivo after LNC transfer prevented paralysis, suggesting that endogenous **IL-12** plays a pivotal role in the pathogenesis of this model of autoimmune disease.

L9 ANSWER 19 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 12

AN 1993:343704 BIOSIS

DN PREV199396040704

TI Resolution of cutaneous leishmaniasis: Interleukin 12 initiates a protective T helper type 1 immune response.

AU Sypek, Joseph P. (1); Chung, Charles L.; Mayor, Sharon E. H.; Subramanyam, Janaki M.; **Goldman, Samuel L.**; Sieburth, Derek S.; Wolf, Stanley F.; Schaub, Robert G.

CS (1) Dep. Preclin. Biol., Genetics Inst. Inc., 87 Cambridge Park Dr., Cambridge, MA 02140 USA

SO Journal of Experimental Medicine, (1993) Vol. 177, No. 6, pp. 1797-1802. ISSN: 0022-1007.

DT Article

LA English

AB Resistance to Leishmania major in mice is associated with the appearance of distinct T helper type 1 (Th1) and Th2 subsets. T cells from lymph nodes draining cutaneous lesions of resistant mice are primarily interferon y (IFN-gamma)-producing Th1 cells. In contrast, T cells from susceptible mice are principally Th2 cells that generate interleukin 4 (IL-4). Although existing evidence is supportive of a role for IFN-gamma in the generation of Th1 cells, additional factors may be required for a protective response to be maintained. A potential candidate is **IL -12,** a heterodimeric cytokine produced by monocytes and B cells that has multiple effects on T and natural killer cell function, including inducing IFN-gamma production. Using an experimental leishmanial model we have observed that daily intraperitoneal administration at the time of parasite challenge of either 0.33 mu-g **IL-12** (a consecutive 5 d/wk for 5 wk) or 1.0 mu-g **IL-12** per mouse (only a consecutive 5 d) caused a gt 75% reduction in parasite burden at the site of infection, in highly susceptible BALB/c mice. Delay of treatment by 1 wk had less of a protective effect. Concomitant with these protective effects was an increase in IFN-gamma and a decrease in IL-4 production, as measured by enzyme-linked immunosorbent assay of supernatants generated from popliteal lymph node cells stimulated with leishmanial antigen in vitro. The reduction in parasite numbers induced by **IL-12** therapy was still apparent at 10 wk postinfection. In addition, we observed that the administration of a rabbit anti-recombinant murine **IL-12** polyclonal

antibody (200 mu-g i.p. every other day for 25 d) at the time of infection to resistant C57Bl/6 mice exacerbated disease. These effects were accompanied by a shift in IFN-gamma production in vitro by antigen-stimulated lymph node cells indicative of a Th2-like response. These findings suggest that **IL-12** has an important role in initiating a Th1 response and protective immunity.

L9 ANSWER 20 OF 20 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 92231358 EMBASE
DN 1992231358
TI Observations, legends, and conjectures concerning restricted T-cell receptor usage and autoimmune disease.
AU Esch T.; Clark L.; Zhang X.-M.; **Goldman S.**; Heber-Katz E.
CS Wistar Institute, 3601 Spruce Street,Philadelphia, PA 19104, United States
SO Critical Reviews in Immunology, (1991) 11/5 (249-264).
ISSN: 1040-8401 CODEN: CCRIDE
CY United States
DT Journal; General Review
FS 005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB It has become clear over the past few years that a variety of experimental autoimmune conditions are mediated by T cells bearing a highly restricted subset of antigen receptors. This restricted TcR usage raises important questions concerning not only the recognition of autoantigens, but also the pathogenic mechanisms underlying many models of autoimmunity. Furthermore, the extension of these findings in certain cases to human disease has raised the possibility of specific therapeutic immune intervention. In this review, we examine the available data on restricted T-cell receptor usage in autoimmune disorders and explore the interpretations and the theoretical and practical implications of these findings.

=> d his

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                E LEONARD J P/AU
L2          359 S E3-E4
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L3           78 S E2-E9
                E GOLDMAN S/AU
L4         1458 S E3
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L5           70 S E3
L6         2059 S L1-L5
L7          109 S L6 AND (IL-12 OR RA OR ARTHRITIS)
L8           43 S L7 AND (ANTIBOD? OR ANTAGONIST?)
L9           20 DUP REM L8 (23 DUPLICATES REMOVED)

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L10           1 L9 AND P40
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L10  ANSWER 1 OF 1  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN   2001:39565  BIOSIS
DN   PREV200100039565
TI   Myelin oligodendrocyte glycoprotein induced EAE in IL-12
     p35 deficient mice.
AU   Hunter, S. E. (1); Thibodeaux, D. K. (1); Bouchard, P. (1); Leonard,
     J. P. (1)
CS   (1) Genetics Institute, Inc., Cambridge, MA, 02140 USA
SO   FASEB Journal, (April 20, 2000) Vol. 14, No. 6, pp. A1116. print.
     Meeting Info.: Joint Annual Meeting of the American Association of
     Immunologists and the Clinical Immunology Society Seattle, Washington, USA
     May 12-16, 2000
     ISSN: 0892-6638.
DT   Conference
LA   English
SL   English


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FULL ESTIMATED COST                                      90.65      91.49

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
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CA SUBSCRIBER PRICE                                      -0.62      -0.62

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FILE CONTAINS CURRENT INFORMATION.
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L3           78 S E2-E9
                E GOLDMAN S/AU
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                E OHARA R/AU
L5           70 S E3
L6         2059 S L1-L5
L7          109 S L6 AND (IL-12 OR RA OR ARTHRITIS)
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L8             43 S L7 AND (ANTIBOD? OR ANTAGONIST?)
L9             20 DUP REM L8 (23 DUPLICATES REMOVED)
L10             1 S L9 AND P40

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                0 EPITOP?
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=> s RA or rheumatoid arthritis
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             0 ARTHRITIS
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L12          2 RA OR RHEUMATOID ARTHRITIS

=> file biosis medline agricola embase caba wpids  japio biotechds lifesci caplus
uspatall
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=> s l9 and (fragment? or epitop?)
L13          3 L9 AND (FRAGMENT? OR EPITOP?)

=> d bib ab 1-3

L13  ANSWER 1 OF 3  WPIDS (C) 2002 THOMSON DERWENT
AN   2002-147853 [19]   WPIDS
DNC  C2002-045892
```

TI Composition for modulating immune response, comprises a spore system having a spore and polypeptide, carbohydrate or nucleotide sequence having anti-pathogenic activity.

DC B04 D16

IN **GOLDMAN, S**; LATHROP, S J; LONGCHAMP, P F; WHALEN, R G

PA (MAXY-N) MAXYGEN INC

CYC 96

PI WO 2002000232 A2 20020103 (200219)* EN 137p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001073009 A 20020108 (200235)

ADT WO 2002000232 A2 WO 2001-US20372 20010626; AU 2001073009 A AU 2001-73009 20010626

FDT AU 2001073009 A Based on WO 200200232

PRAI US 2000-214161P 20000626

AB WO 200200232 A UPAB: 20020321
NOVELTY - A composition (I), comprising a spore system (II) having a spore and a peptide, polypeptide, protein, carbohydrate or nucleotide sequence having anti-pathogenic activity displayed on, bound to or contained within, the spore, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) releasing a spore system of interest, comprising:

(a) transforming a cell capable of sporulation with an exogenous nucleic acid;

(b) inducing sporulation of the cell, where at least one spore system is produced; and

(c) lysing the cell to release the spore system;

(2) displaying a polypeptide at one or more sites of interest on a surface of a spore, comprising:

(a) transforming a cell capable of sporulation with a recombinant nucleic acid vector, comprising a nucleic acid encoding a polypeptide fused in frame to a nucleic acid encoding a spore protein; and

(b) expressing a fusion protein comprising the polypeptide and the spore coat protein so that the fusion protein is attached to the spore coat of the spore at one or more site of interest on the spore surface;

(3) a detection system (DS) comprising (II) which comprises a moiety that provides a detectable signal and a polypeptide capable of capturing a detectable compound;

(4) delivery of a polypeptide of interest, comprising:

(a) transforming a cell that is capable of sporulating with a nucleic acid encoding the polypeptide;

(b) inducing sporulation of the cell to form a spore; and

(c) delivering the spore to a site of interest;

(5) modulation of an adjuvant effect in an organism, comprising:

(a) generating a non-viable spore, having an adjuvant effect;

(b) isolating the spore; and

(c) contacting the organism with the spore and a nucleic acid, polypeptide, or peptide; and

(6) enhancing (M1) an immune response to an immunogenic polypeptide or peptide in a subject, comprising administering (I).

ACTIVITY - Antibacterial; virucide; anti-HIV (human immunodeficiency virus); cytostatic; neuroprotective; nootropic; hepatotropic; antipyretic; antiinflammatory; antirheumatic; antiarthritic; antidiabetic; immunosuppressive; antipsoriatic; antiallergic; antiasthmatic.

MECHANISM OF ACTION - Modulator of immune response (claimed); vaccine.

Spores from Bacillus subtilis were tested to determine if the spores

had an adjuvant effect. The specific immunological response of mice to spores and V-antigen mixed together was compared to the specific immunological response of mice to the V-antigen protein alone. 1 micro g, 0.5 micro g or 0.25 micro g of purified recombinant V-antigen was mixed with 5 multiply 108 non-recombinant B. subtilis spores or used alone. The three V-antigen protein/spore mixtures and three amounts of V-antigen protein were injected intraperitoneally into separate groups of mice, at days 1, 21 and 35. Mice were bled on days 10, 21 and 45. Serum was analyzed for specific and V-antigen immunoglobulins by an indirect enzyme linked immunosorbent assay (ELISA) using standard procedures. The presence of spores in the inoculum increased the **antibody** titer between 10-fold and 1000-fold, depending on the amount of protein inoculated. The data suggested that spores act to augment a specific immune response to immunogenic polypeptide, such as V-antigen protein.

USE - (I) is useful for modulating (producing or enhancing) an immune response of an organism, and for generating a desired product. DS is useful for detecting a compound. (All claimed). The spores are useful in production, packaging, delivering and presentation systems for industrial biocatalyst and in medical applications including immunization and vaccination. The spores are also useful as therapeutics and/or prophylactic agents, and as vaccines against a broad spectrum of immunogens and bacterial, viral and parasitic pathogens and toxins. The spores are also useful for production and immobilization of enzymes and proteins for industrial use, and in a variety of biotechnology settings as carriers for nucleic acids and biotin linked ligands. (II) is useful as sensor and detector. (II) is useful as a vaccine or immunomodulatory agent against a disease or disease causing pathogen including Staphylococcus sp., Streptococcus sp., viral encephalitis, human immunodeficiency virus (HIV), cytomegalovirus, poliomyelitus, rabies, cancer, typhoid, parasites, anthrax, foot and mouth disease, Alzheimer's disease, hepatitis, diphtheria, pertussis, hemorrhagic fevers, influenza, cholera, meningitis, measles, mumps, Lyme disease, tetanus, yellow fever and pneumonia. (I) is also useful for treating allergy, asthma, autoimmune diseases, e.g. rheumatoid **arthritis**, diabetes mellitus and multiple sclerosis, septic shock, organ transplantation and inflammatory conditions including inflammatory bowel syndrome, psoriasis, pancreatitis, and other immunodeficiencies.

Dwg.0/12

L13 ANSWER 2 OF 3 WPIDS (C) 2002 THOMSON DERWENT
AN 2000-282570 [24] WPIDS
CR 1997-132638 [12]; 1997-165283 [15]; 2000-430395 [36]
DNC C2000-085202
TI Novel human CTLA-8 protein useful for treating immunodeficiencies and disorders, in regulating growth, proliferation and/or activity of T and/or B lymphocytes and multiple sclerosis, rheumatoid **arthritis**.
DC B04 D16
IN CARLIN, M; GIANNOTTI, J; GOLDEN-FLEET, M M; **GOLDMAN**, **S**; JACOBS, K; KELLEHER, K; MI, S; NEBEN, S; PITTMAN, D
PA (GEMY) GENETICS INST INC
CYC 1
PI US 6043344 A 20000328 (200024)* 25p
ADT US 6043344 A Provisional US 1995-35347P 19950719, CIP of US 1995-504032 19950719, CIP of US 1995-514014 19950811, Div ex US 1996-685239 19960718, US 1998-34810 19980304
FDT US 6043344 A CIP of US 5707829
PRAI US 1995-35347P 19950719; US 1995-504032 19950719; US 1995-514014 19950811; US 1996-685239 19960718; US 1998-34810 19980304
AB US 6043344 A UPAB: 20010711
NOVELTY - An isolated human CTLA-8 (B18) (I) with a fully defined sequence as given in the specification, is new.

DETAILED DESCRIPTION - (I) comprises a fully defined sequence of 163

(2) amino acids, amino acids 11-, 29- or 31-163 of (2), the **fragment** of (2) comprising amino acids 11-, 29- or 31-163 of (2), as given in the specification.

An INDEPENDENT CLAIM is also included for the preparation of (I).

ACTIVITY - Immunosuppressive; antiarthritic; antiinflammatory; immunostimulant; antidiabetic; neuroprotective; dermatological; antianemic; antiallergic; antithyroid; antiasthmatic; antibacterial; cytostatic.

MECHANISM OF ACTION - Angiogenesis inhibitor; hematopoiesis regulator; growth or proliferation of vascular endothelial cells inhibitor; tumor growth inhibitor; myeloid, lymphoid cells or their progenitors proliferator; IFN- gamma , IL-3, GM-CSF production inducer; gene therapy. The ability of (I) to inhibit angiogenesis was examined in an angiostatic activity assay. Primary human umbilical cells (HUVECs) were seeded to 2 multiply 103 cells/well of a 96 well plate and incubated. The cells were then starved in M199 medium. Conditioned media containing B18 was obtained from transfected COS or stably expressing CHO cells and 1:10, 1:50, 1:250 and 1:1250 were prepared in M199-CS medium containing 100 ng/ml FGF. The dilutions of B18 were added to the starved cells and incubated for 72 hr at 37 deg. C. The cells were then radiolabeled and trypsinized for liquid scintillation counting, after washing. Results showed that human CTLA-8 (B18) inhibits angiogenesis.

USE - (I) is used for treating immune deficiencies and disorders (including severe combined immunodeficiency (SCID), e.g. in regulating growth and proliferation of T and/or B lymphocytes, and effecting the cytolytic activity of NK cells and other cell populations. These immune deficiencies may be genetic or caused by viruses, bacterial or fungal infections. The proteins are also used for boosting the immune system for treating cancer and in the treatment of autoimmune diseases such as multiple sclerosis, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent diabetes mellitis, myasthenia gravis, graft-versus-host disease and autoimmune inflammatory eye disease. They are also used for treating asthma, allergic reactions or other respiratory problems and suppressing chronic or acute inflammation associated with infection such as septic shock or systemic inflammatory response syndrome (SIRS), inflammatory bowel disease and Crohn's disease. (I) is also used for regulating hematopoiesis and consequently in the treatment of myeloid or lymphoid deficiencies i.e. by supporting the growth and proliferation of erythroid progenitor cells, myeloid cells, megakaryocytes, hematopoietic stem cells and thus used for treating anemia, thrombocytopenia, aplastic anemia and paroxysmal nocturnal hemoglobinuria. They also inhibit the growth and proliferation of vascular endothelial cells and thus are effective in inhibiting angiogenesis. The polynucleotides encoding (I) can be used in gene therapy. The proteins are used as immunogens to produce polyclonal or monoclonal **antibodies** useful for performing diagnostic assays for CTLA-8.

DESCRIPTION OF DRAWING(S) - The figure shows the data relating to the ability of CTLA-8 to inhibit angiogenesis.

Dwg.3/7

L13 ANSWER 3 OF 3 WPIDS (C) 2002 THOMSON DERWENT
AN 1997-132638 [12] WPIDS
CR 1997-165283 [15]; 2000-282570 [23]; 2000-430395 [36]
DNC C1997-042879
TI New nucleic acid encoding the CTLA-8 protein - modulates growth of vascular endothelial and haematopoietic cells and induces cytokine expression, for treating infection, auto-immune disease, etc..
DC B04 D16
IN CARLIN, M; JACOBS, K; KELLEHER, K; MCCOY, J M; GIANNOTTI, J; GOLDEN-FLEET, M; **GOLDMAN**, **S**; MI, S; NEBEN, S; PITTMAN, D; DUCKETT, M C; GOLDEN-FLEET, M M; PITMAN, D; CARLIN-DUCKETT, M

PA (GEMY) GENETICS INST INC
CYC 23
PI WO 9704097 A2 19970206 (199712)* EN 50p
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AU CA JP MX
AU 9667123 A 19970218 (199723)
WO 9704097 A3 19970912 (199749)
US 5707829 A 19980113 (199809) 30p
EP 839196 A2 19980506 (199822) EN
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
JP 11510045 W 19990907 (199947) 59p
US 5969093 A 19991019 (199950)
MX 9800507 A1 19980501 (200007)
MX 9801120 A1 19990401 (200055)
AU 727480 B 20001214 (200103)
AU 727489 B 20001214 (200103)
AU 2001028001 A 20010517 (200138)#
AU 2001028002 A 20010802 (200152)#
ADT WO 9704097 A2 WO 1996-US11889 19960718; AU 9667123 A AU 1996-67123 19960218; US 5707829 A US 1995-514014 19950811; EP 839196 A2 EP 1996-927237 19960718, WO 1996-US11889 19960718; JP 11510045 W WO 1996-US11889 19960718, JP 1997-506846 19960718; US 5969093 A Div ex US 1995-514014 19950811, US 1997-833823 19970410; MX 9800507 A1 MX 1998-507 19980116; MX 9801120 A1 MX 1998-1120 19980210; AU 727480 B AU 1996-67123 19960718; AU 727489 B AU 1996-67685 19960808; AU 2001028001 A Div ex AU 1996-67685 19960808, AU 2001-28001 20010314; AU 2001028002 A Div ex AU 1996-67123 19960718, AU 2001-28002 20010314
FDT AU 9667123 A Based on WO 9704097; EP 839196 A2 Based on WO 9704097; JP 11510045 W Based on WO 9704097; AU 727480 B Previous Publ. AU 9667123, Based on WO 9704097; AU 727489 B Previous Publ. AU 9667685, Based on WO 9707198; AU 2001028001 A Div ex AU 727489; AU 2001028002 A Div ex AU 727480
PRAI US 1995-514014 19950811; US 1995-504032 19950719; US 1997-833823 19970410; WO 1996-US12897 19960808; AU 2001-28001 20010314; AU 2001-28002 20010314
AB WO 9704097 A UPAB: 20011001
A novel isolated polynucleotide (I) comprises: (a) nucleotides (nt) 146-544 of an 813 bp sequence given in the specification; (b) a sequence able to hybridise with (a) or varying from (a) only within the degeneracy of the genetic code; or (c) an allelic variant of (a). Also claimed are: (1) host cells transformed with (I); (2) isolated human CTLA-8 protein which has 163 amino acids (aa), its 11-163, 29-163 or 31-163 regions or any **fragments** of them with CTLA-8 activity; and (3) **antibodies** (Ab) which specifically react with CTLA-8 protein.

USE - (I) encodes proteins with CTLA-8 activity. Treatment of mammals with CTLA-8 (or non-human analogues or IL-17) results in at least one of: (a) inhibition of angiogenesis, growth/proliferation of vascular endothelial cells, tumour cells and angiogenesis-dependent tissue growth; (b) proliferation of myeloid, erythroid or lymphoid cells (or their progeny); or (c) induction of interferon- gamma , IL-3 or GM-CSF prodn (claimed). Opt. CTLA-8 is expressed in vivo from a suitable vector. Typical applications of CTLA-8 are treatment of immune deficiency and disorders requiring modulation of T/B cell growth or proliferation, or of cytolytic natural killer cells, e.g. viral or microbial infection (e.g. HIV, hepatitis, malaria, candidiasis etc.); autoimmune disease (e.g. multiple sclerosis, rheumatoid **arthritis**, insulin-dependent diabetes etc.); to boost the immune response in cancer treatment; as antiinflammatories (e.g. in septic shock or Crohn's disease) and in haematopoietic disorders where growth/proliferation of erythroid, myeloid or megakaryocytic cells is needed. Ab can be used to determine CTLA-8, possibly also for treating some tumours or some of the above conditions. Dwg.0/7

p40 production by anti-CD3 was abrogated by anti-CD154 **antibody**. **IL-12 p40** production was also increased by LPS stimulation. LPS-stimulated **IL-12** production was inhibited by anti-TNFalpha **antibody**, but not by T cell depletion and anti-CD154 **antibody** treatment. The TNFalpha inhibitor rolipram inhibited LPS-stimulated **IL-12 p40** production by **RA** SC more strongly than spontaneous production. TNFalpha restored LPS-stimulated **IL-12** production that had been inhibited by rolipram. Conclusion: **IL-12** production in **RA** is regulated by 2 different pathways. One pathway is T cell dependent, predominantly through a CD40-CD154 interaction, while the other is T cell independent, mediated through TNFalpha. Inhibition of **IL-12** production by interference with CD40-CD154 interaction and TNFalpha production may be a potential therapeutic strategy for treating **RA**.

L19 ANSWER 2 OF 18 WPIDS (C) 2002 THOMSON DERWENT
AN 2001-244560 [25] WPIDS
DNC C2001-073385
TI Composition comprising interleukin-12 **p40** and IL-B30 polypeptide or its segment, useful for ameliorating **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor.
DC B04 D16
IN DE WAAL MALEFYT, R; KASTELEIN, R A; LIRA, S A; NARULA, S K; OPPMANN, B; RENNICK, D M; WIEKOWSKI, M T
PA (SCHE) SCHERING CORP
CYC 92
PI WO 2001018051 A2 20010315 (200125)* EN 69p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CZ DE DK DM DZ EE ES FI GB GD GE HR HU ID IL IN IS JP KG KR KZ LC LK LR LT LU LV MA MD MG MK MN MX MZ NO NZ PL PT RO RU SE SG SI SK SL TJ TM TR TT TZ UA UZ VN YU ZA
AU 2000073608 A 20010410 (200137)
EP 1210434 A2 20020605 (200238) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
ADT WO 2001018051 A2 WO 2000-US24686 20000908; AU 2000073608 A AU 2000-73608 20000908; EP 1210434 A2 EP 2000-961688 20000908, WO 2000-US24686 20000908
FDT AU 2000073608 A Based on WO 200118051; EP 1210434 A2 Based on WO 200118051
PRAI US 1999-164616P 19991110; US 1999-393090 19990909
AB WO 200118051 A UPAB: 20010508
NOVELTY - A composition (I) comprising a substantially pure polypeptide comprising a number of distinct segments of at least 7 contiguous amino acids from interleukin (**IL**)-**12 p40** and/or IL-B30, and a substantially pure polypeptide comprising a segment of at least 11 contiguous amino acids from **IL-12 p40** and/or IL-B30.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated or recombinant nucleic acid (II) encoding (I);

(2) a cell (III) comprising (II);

(3) a nucleic acid (IV) which hybridizes under wash conditions of 30 minutes at 50 deg. C and less than 1M salt to the natural mature coding portion of primate **IL-12 p40** and IL-B30;

(4) an **antagonist** (V) of **IL-12 p40**/IL-B30 combined with a tumor necrosis factor-alpha (TNF alpha) **antagonist**, an **IL-12 antagonist**, IL-10, or steroids;

(5) a binding compound (VI) comprising an antigen binding site from

an **antibody**, which specifically binds to (I) and comprising a substantially pure polypeptide comprising **IL-12 p40** and IL-B30 polypeptide, or a polypeptide comprising **IL-12 p40** fused to IL-B30, but not to either **IL-12 p40** or IL-B30 polypeptide;

(6) a kit (VII) comprising:

(a) (I), and a compartment comprising the polypeptide, or instructions for use or disposal of reagents in the kit;

(b) (II), and a compartment comprising (II), a compartment further comprising a primate **IL-12 p40** or IL-B30, or instructions for use or disposal of reagents in the kit or (VI); and

(c) a compartment comprising (VI), or instructions for use or disposal of reagents in the kit;

(7) producing (M1) an antigen:**antibody** complex, involves contacting, under appropriate conditions, a primate **IL-12 p40**/IL-B30 composition with (VI), allowing the complex to form;

(8) a composition (VIII) comprising (VI) which is sterile, or (VI) and a carrier such as an aqueous compound, including water, saline, and/or buffer;

(9) increasing (M2) the secretion of a primate IL-B30 involves expressing the polypeptide with **IL-12 p40** or increasing the secretion of a primate **IL-12 p40** involves expressing the **IL-12 p40** with IL-B30; and

(10) screening (M3) for a receptor which binds (I) involves contacting the complex to a cell expressing the receptor under conditions allowing the complex to bind to the receptor, forming a detectable interaction.

ACTIVITY - Antirheumatic; antiarthritic; osteopathic; antiarthritic; neuroprotective; antiarteriosclerotic; cerebroprotective; vasotropic; cytostatic; antitumor; immunosuppressive.

MECHANISM OF ACTION - Modulator of physiology or development of cell in host; inducer of memory T-cell proliferation (claimed); modulator of trafficking or activation of leukocyte.

No supporting data is given.

USE - (I) is useful for modulating physiology or development of a cell or tissue in a host organism by contacting the cell with (I) or (V), resulting in an increased or decreased production of Interferon-gamma (IFN gamma), an enhanced Th1 response such as anti-tumor effect, adjuvant effect, anti-viral effect or antagonized allergic effect, and amelioration of an autoimmune condition or a chronic inflammatory condition. The contacting is in combination with IL-18, **IL-12**, radiation therapy or chemotherapy, an immune adjuvant or an anti-viral therapeutic. The **antagonist** is an **antibody** against **IL-12** receptor subunit beta 1. The **antagonist** or agonist of mammalian IL-B30 protein is useful for modulating the inflammatory response in an animal, by contacting cells in the animal with the agonist or **antagonist**, where the animal exhibits signs or symptoms of an acute phase inflammatory response in skin, lung, gastrointestinal, or liver tissue. The modulation is accelerating maturation of neutrophils into platelets and has an effect on immunoglobin A and G (IgA and IgG) . The **antagonist** is an **antibody** which binds to the mammalian IL-B30 or blocks signaling mediated by mammalian IL-B30. The **antagonist** or agonist is administered in combination with an anti-inflammatory cytokine agonist or **antagonist**, an analgesic, an anti-inflammatory agent, or a steriod. IL-B30 or its agonist is useful inducing the proliferation of memory T-cells (all claimed).

Agonist or **antagonist** of IL-B30 protein is useful for modulating the trafficking or activation of a leukocyte in an animal experiencing science or symptoms of autoimmunity, an inflammatory

or heteroaryl; each of R.sub.2, R.sub.4, and R.sub.5, independently, is R.sup.c, halogen, nitro, nitroso, cyano, azide, isothionitro, SR.sup.c, or OR.sup.c; R.sub.3 is R.sup.c, alkenyl, alkynyl, aryl, heteroaryl, cyclyl, heterocyclyl, OR.sup.c, OC(O)R.sup.c, SO.sub.2R.sup.c, S(O)R.sup.c, S(O.sub.2)NR.sup.cR.sup.d, SR.sup.c, NR.sup.cR.sup.d, NR.sup.cCOR.sup.d, NR.sup.cC(O)OR.sup.d, NR.sup.cC(O)NR.sup.cR.sup.d, NR.sup.cSO.sub.2R.sup.d, COR.sup.c, C(O)OR.sup.c, or C(O)NR.sup.cR.sup.d; n is 0, 1, 2, 3, 4, 5, 6, or 7; X is O, S, S(O), S(O.sub.2), or NR.sup.c; Y is a covalent bond, CH.sub.2, C(O), C.dbd.N--R.sup.c, C.dbd.N--OR.sup.c, C.dbd.N--SR.sup.c, O, S, S(O), or S(O.sub.2); Z is N; and W is O, S, S(O), S(O.sub.2), NR.sup.c, or NC(O)R.sup.c; in which each of R.sup.a and R.sup.b, independently, is H, alkyl, aryl, heteroaryl; and each of R.sup.c and R.sup.d, independently, is H, alkyl, or alkylcarbonyl.

L19 ANSWER 5 OF 18 USPATFULL
AN 2001:229210 USPATFULL
TI Methods for enhancing oral tolerance and treating autoimmune disease using inhibitors of interleukin-12
IN Strober, Warren, Bethesda, MD, United States
Kelsall, Brian, Washington, DC, United States
Marth, Thomas, Kensington, MD, United States
PA Government of the United States of America, Department of Health and Human Services (U.S. corporation)
PI US 2001051159 A1 20011213
AI US 2000-732502 A1 20001207 (9)
RLI Continuation of Ser. No. US 1999-284169, filed on 9 Apr 1999, ABANDONED A 371 of International Ser. No. WO 1996-US16007, filed on 11 Oct 1996, UNKNOWN
DT Utility
FS APPLICATION
LREP mary l. miller THE CANDLER BUILDING, needle & rosenberg, p.c., 127 peachtree street, n.e., atlanta, GA, 30303-1811
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1252
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a method for enhancing oral tolerance to an antigen associated with an autoimmune disease in a subject having the autoimmune disease comprising orally administering to the subject an antigen associated with the autoimmune disease and administering an inhibitor of interleukin-12 in amounts sufficient to enhance oral tolerance. Also provided in the present invention is a method for treating or preventing an autoimmune disease in a subject comprising orally administering to the subject an antigen associated with the autoimmune disease and administering an inhibitor of interleukin-12 in amounts sufficient to treat or prevent the autoimmune disease, thereby treating or preventing the autoimmune disease.

L19 ANSWER 6 OF 18 USPATFULL
AN 2001:221075 USPATFULL
TI Retinoid antagonists and use thereof
IN Bollag, Werner, Basel, Switzerland
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of
Mohr, Peter, Basel, Switzerland
Panina-Bordignon, Paola, Milan, Italy
Rosenberger, Michael, Caldwell, NJ, United States
Sinigaglia, Francesco, Milan, Italy
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6326397 B1 20011204

AI US 1999-307009 19990507 (9)
RLI Continuation-in-part of Ser. No. US 1998-189189, filed on 10 Nov 1998
DT Utility
FS GRANTED
EXNAM Primary Examiner: Killos, Paul J.
LREP Johnston, George W., Parise, John P.
CLMN Number of Claims: 16
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 1573
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel retinoid antagonists of the formula I ##STR1##

wherein the dotted bond can be either hydrogenated or form a double bond; and, when the dotted bond forms a double bond, R.sup.1 is lower alkyl and R.sup.2 is hydrogen; and, when the dotted bond is hydrogenated, R.sup.1 and R.sup.2 taken together are methylene to form a cis-substituted cyclopropyl ring; R.sup.3 is hydroxy or lower alkoxy; R.sup.4 is alkyl or alkoxy; and R.sup.5 and R.sup.6 are, independently, a C.sub.4-12 alkyl or a 5-12 cycloalkyl substituent containing from 1-3 rings which are either unsubstituted or substituted with from 1-3 lower alkyl groups, with the carbon atom of R.sup.5 and R.sup.6 being linked to the remainder of the molecule to form a quaternary carbon atom pharmaceutically acceptable salts of carbocylic acids of the formula I; as well as method for the treatment of osteoporosis and preneoplastic and neoplastic diseases, and a method for reducing or abolishing adverse events in subjects receiving retinoid agonist treatment by administering a retinoid **antagonist**.

L19 ANSWER 7 OF 18 USPATFULL
AN 2001:63494 USPATFULL
TI **Antibodies** against human **IL-12**
IN Gately, Maurice Kent, Parsippany, NJ, United States
Presky, David Howard, Glen Ridge, NJ, United States
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6225117 B1 20010501
AI US 1999-232522 19990119 (9)
PRAI US 1998-72333P 19980123 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: DiBrino, Marianne
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1122
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel p75 heterodimer specific anti-human **IL-12 antibodies** that are characterized by a higher potency and greater efficacy in neutralizing human **IL-12** bioactivity than known heterodimer specific **IL-12** monoclonal **antibodies**. The heterodimer specific **antibodies** recognize one or more **epitopes** of the human **IL-12** p75 heterodimer, but do not bind to the **p40** subunit alone. The heterodimer specific **IL-12 antibodies** neutralize rhesus monkey **IL-12** bioactivity with a potency similar to their potency for neutralizing human **IL-12** bioactivity making them useful **IL-12** antagonists for in vivo studies in the rhesus monkey.

L19 ANSWER 8 OF 18 USPATFULL
AN 2000:138395 USPATFULL
TI Treatment of T-helper cell type 2-mediated immune disease by retinoid antagonists
IN Bollag, Werner, Basel, Switzerland
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of
Panina-Bordignon, Paola, Milan, Italy
Sinigaglia, Francesco, Milan, Italy
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6133309 20001017
AI US 1998-189189 19981110 (9)
PRAI EP 1997-119776 19971112
DT Utility
FS Granted
EXNAM Primary Examiner: Travers, Russell
LREP Johnston, George W., Epstein, William H., Parise, John P.
CLMN Number of Claims: 37
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 780
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Retinoids with retinoid receptor antagonistic activity, pharmaceutically acceptable salts and pharmaceutically acceptable hydrolyzable esters thereof, have been found efficacious in treating T-helper cell type 2 (Th2)-mediated immune diseases, such as immunoglobulin E (IgE)-mediated allergic diseases.

L19 ANSWER 9 OF 18 USPATFULL
AN 2000:98551 USPATFULL
TI Treatment of papillomavirus-associated lesions
IN Stanley, Margaret Anne, Cambridge, United Kingdom
Scarpini, Cinzia Giuseppina, Cambridge, United Kingdom
PA Cambridge University Technical Services, Ltd., Cambridge, United Kingdom (non-U.S. corporation)
PI US 6096869 20000801
AI US 1996-621841 19960322 (8)
PRAI GB 1995-5784 19950322
DT Utility
FS Granted
EXNAM Primary Examiner: Park, Hankyel
LREP Klarquist Sparkman Campbell Leigh & Whinston, LLP
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1293
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Interleukin-12 (**IL-12**) or a functional analogue thereof, or a polynucleotide encoding **IL-12** or encoding a functional analogue thereof, is used as a therapeutic material or adjuvant in treating papillomavirus-associated lesions e.g. warts due to HPV 6 and/or 11, e.g. condyloma acuminata. **IL-12** or a vector encoding it for endogenous production can be used together with a vaccine such as a papillomavirus antigen, or a vector encoding a papillomavirus antigen.

L19 ANSWER 10 OF 18 USPATFULL
AN 2000:87729 USPATFULL
TI Method of converting a Th2-type allergic immune response into a Th1-type immune response
IN DeKruyff, Rosemarie H., Stanford, CA, United States
Umetsu, Dale T., Stanford, CA, United States

PA The Board of Trustees of the Leland Stanford Junior University, Palo Alto, CA, United States (U.S. corporation)
PI US 6086898 20000711
AI US 1999-339068 19990623 (9)
PRAI US 1998-90390P 19980623 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: Ewoldt, Gerald R.
LREP Bozicevic, Field & Francis, Sherwood, Pamela
CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN 17 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 1767
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods are provided for the treatment of allergic and other immune disorders associated with overproduction of Th2 type cytokines by antigen specific T cells. Immunotherapy with adjuvants, as provided in the present invention, greatly inhibits the development of airway hyperreactivity and airway inflammation. Such immunotherapy is shown to reverse ongoing airway disease, and convert allergic inflammatory responses into protective immune responses. Conditions of particular interest include allergic conditions associated with production of Th2 cytokines and/or IgE **antibodies**, asthma, allergic rhinitis, and anaphylactic reactions. The addition of adjuvant induces a Th1-type immune response and can redirect an established Th2-type response to a Th1-type response for the selected antigen. Preferably, antigen-specific IgE production is reduced without altering the intensity of the antigen-specific proliferative response. One particularly preferred adjuvant for use in accordance with the present invention is a Listeria adjuvant.

L19 ANSWER 11 OF 18 USPATFULL
AN 2000:87707 USPATFULL
TI Methods and compositions for the inhibition of interleukin-12 production
IN Karp, Christopher L., Lutherville, MD, United States
Trinchieri, Giorgio, Wynnewood, PA, United States
Wysocka, Maria, Wynnewood, PA, United States
Griffin, Diane E., Hunt Valley, MD, United States
PA The Wistar Insitute, Philadelphia, PA, United States (U.S. corporation)
Johns Hopkins University, Baltimore, MD, United States (U.S. corporation)
PI US 6086876 20000711
AI US 1998-19862 19980206 (9)
PRAI US 1997-37722P 19970207 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Kemmerer, Elizabeth; Assistant Examiner: Romeo, David S.
LREP Akin, Gump, Strauss, Hauer & Feld, L.L.P.
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 13 Drawing Figure(s); 18 Drawing Page(s)
LN.CNT 1487
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention includes compositions and methods for selective suppression of **IL-12** production in a cell. Methods of treating a human having a disease associated with dysregulated **IL-12** production are also provided.

L19 ANSWER 12 OF 18 USPATFULL
AN 2000:50737 USPATFULL

formula ##STR1## wherein A is a single or double bond,

B.sup.1 and B.sup.2 are each independently CH.dbd.CH or C.tbd.C,

T is CH.sub.2 or CH.sub.2 CH.sub.2,

X is --CH.sub.2 -- or >C.dbd.CH.sub.2,

R.sup.1 is H, F or OH,

R.sup.2 and R.sup.3 are each independently lower alkyl or CF.sub.3, or

C(R.sup.2,R.sup.3) is C.sub.3-6 -cycloalkyl,

are useful in the treatment or prevention of vitamin D dependent disorders and of **IL-12**-dependent autoimmune diseases, particularly psoriasis, basal cell carcinomas, disorders of keratinization and keratosis, leukemia, osteoporosis, hyperparathyroidism accompanying renal failure, multiple sclerosis, transplant rejection, graft vs. host disease, **rheumatoid arthritis**, insulin-dependent diabetes mellitus, inflammatory bowel disease, septic shock and allergic encephalomyelitis.

L19 ANSWER 14 OF 18 USPATFULL
AN 1999:75759 USPATFULL
TI Low affinity human **IL-12** beta2 receptor
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States
Presky, David Howard, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5919903 19990706
AI US 1997-914520 19970819 (8)
RLI Division of Ser. No. US 1996-685118, filed on 23 Jul 1996
PRAI US 1995-1701P 19950801 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 2
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1531
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A recombinant human **IL-12** receptor complex produced on the surface of a non-human mammalian cell and free from other human proteins, the complex comprising the beta1 receptor protein complexed with a beta2 receptor protein, which complex is capable of binding to human **IL-12** with high affinity. A recombinant human **IL-12** beta2 receptor protein produced on the surface of a non-human mammalian cell, free from other human proteins, in its active form. In addition, a non-human mammalian cell having expressed on its surface the recombinant human **IL-12** beta2 receptor protein or the recombinant human **IL-12** receptor complex, which cell proliferates in the presence of human **IL-12**. A non-human mammalian cell having the human **IL-12** beta2 receptor protein or the complex expressed on its surface and which proliferates in response to human **IL-12** is useful for determining whether a given compound inhibits biological activity of human **IL-12** or is an **IL-12** agonist.

L19 ANSWER 15 OF 18 USPATFULL
AN 1998:160106 USPATFULL

TI **Antibodies** to receptors for human interleukin-12
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States
Presky, David Howard, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5852176 19981222
AI US 1997-915495 19970820 (8)
RLI Division of Ser. No. US 1996-685118, filed on 23 Jul 1996
PRAI US 1995-1701P 19950801 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 1
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1381
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB **Antibodies** to human **IL-12** beta 2 receptor protein or an **IL-12** receptor complex, the complex comprising the beta1 receptor protein complexed with a beta2 receptor protein, which complex is capable of binding to human **IL-12** with high affinity.

L19 ANSWER 16 OF 18 USPATFULL
AN 1998:147252 USPATFULL
TI DNA encoding receptors for the beta-2 chain of human **IL-12**
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States
Presky, David Howard, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5840530 19981124
AI US 1996-685118 19960723 (8)
PRAI US 1995-1701P 19950801 (60)
US 1996-18674P 19960530 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1424
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A recombinant human **IL-12** beta2 receptor protein produced on the surface of a non-human mammalian cell, free from other human proteins, in its active form. In addition, a non-human mammalian cell having expressed on its surface the recombinant human **IL-12** beta2 receptor protein, which cell proliferates in the presence of human **IL-12**. A non-human mammalian cell having the human **IL-12** beta2 receptor protein on its surface and which proliferates in response to human **IL-12** is useful for determining whether a given compound inhibits biological activity of human **IL-12** or is an **IL-12** agonist.

L19 ANSWER 17 OF 18 USPATFULL
AN 1998:135151 USPATFULL
TI Human receptor for interleukin-12
IN Chua, Anne On, Wayne, NJ, United States
Gubler, Ulrich Andreas, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5831007 19981103

AI US 1995-419652 19950411 (8)
RLI Division of Ser. No. US 1994-248532, filed on 31 May 1994, now patented, Pat. No. US 5536657 which is a continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John
LREP Johnston, George W., Epstein, William H., Bucholz, Briana C.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 35 Drawing Figure(s); 26 Drawing Page(s)
LN.CNT 1937
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to substantially pure Interleukin-12 receptor cDNAs and protein and uses therefore. The Interleukin-12 receptor is shown to be a member of the cytokine receptor superfamily and has a high homology to human gp130.

L19 ANSWER 18 OF 18 USPATFULL
AN 96:63048 USPATFULL
TI Recombinant DNA encoding human receptor for interleukin-12
IN Chua, Anne O., Wayne, NJ, United States
Gubler, Ulrich A., Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5536657 19960716
AI US 1994-248532 19940531 (8)
RLI Continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John
LREP Gould, George M., Johnston, George W., Kass, Alan P.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 34 Drawing Figure(s); 25 Drawing Page(s)
LN.CNT 1755
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to substantially pure Interleukin-12 receptor cDNAs and protein and uses therefore. The Interleukin-12 receptor is shown to be a member of the cytokine receptor superfamily and has a high homology to human gp130.

=> d his

(FILE 'HOME' ENTERED AT 11:40:44 ON 11 JUL 2002)

FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABA, WPIDS, JAPIO, BIOTECHDS, LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:42:50 ON 11 JUL 2002
E LEONARD JOHN P/AU
L1 108 S E3-E5
E LEONARD J P/AU
L2 359 S E3-E4
E GOLDMAN SAMUEL/AU
L3 78 S E2-E9
E GOLDMAN S/AU
L4 1458 S E3
E OHARA RICHARD/AU
E OHARA R/AU
L5 70 S E3
L6 2059 S L1-L5
L7 109 S L6 AND (IL-12 OR RA OR ARTHRITIS)

```
L8           43 S L7 AND (ANTIBOD? OR ANTAGONIST?)
L9           20 DUP REM L8 (23 DUPLICATES REMOVED)
L10           1 S L9 AND P40

     FILE 'STNGUIDE' ENTERED AT 11:51:09 ON 11 JUL 2002
L11           0 S L8 AND (FRAGMENT? OR EPITOP?)
L12           2 S RA OR RHEUMATOID ARTHRITIS

     FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABA, WPIDS, JAPIO, BIOTECHDS,
     LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:55:50 ON 11 JUL 2002
L13           3 S L9 AND (FRAGMENT? OR EPITOP?)
L14      656173 S RA OR RHEUMATOID ARTHRITIS
L15        1079 S L14 AND IL-12
L16         724 S L15 AND (ANTIBOD? OR EPITOP? OR FRAGMENT? OR ANTAGONIST)
L17          95 S L16 AND IL-12 (5A) ANTIBOD?
L18          82 DUP REM L17 (13 DUPLICATES REMOVED)
L19          18 S L18 AND P40

=> s l16 and IL-12 (5a) antagonist?
L20          52 L16 AND IL-12 (5A) ANTAGONIST?

=> dup rem l20
PROCESSING COMPLETED FOR L20
L21          44 DUP REM L20 (8 DUPLICATES REMOVED)

=> d bib ab 1-44

L21  ANSWER 1 OF 44  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE
     1
AN   2002:166945  BIOSIS
DN   PREV200200166945
TI   Use of IL-12 and IL-12
     antagonists in the treatment of autoimmune diseases.
AU   Leonard, John (1); Goldman, Samuel; O'Hara, Richard, Jr.
CS   (1) Auburn, NH USA
     ASSIGNEE: Genetics Institute, Inc.
PI   US 6338848 January 15, 2002
SO   Official Gazette of the United States Patent and Trademark Office Patents,
     (Jan. 15, 2002) Vol. 1254, No. 3, pp. No Pagination.
     http://www.uspto.gov/web/menu/patdata.html. e-file.
     ISSN: 0098-1133.
DT   Patent
LA   English
AB   Method of treating autoimmune conditions are disclosed comprising
     administering to a mammalian subject IL-12 or an
     IL-12 antagonist. In certain preferred
     embodiments the autoimmune condition is one which is promoted by an
     increase in levels of IFN-gamma or TNF-alpha. Suitable conditions for
     treatment include multiple sclerosis, systemic lupus erythematosus,
     rheumatoid arthritis, autoimmune pulmonary inflammation,
     Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent
     diabetes melitis and autoimmune inflammatory eye disease.

L21  ANSWER 2 OF 44  USPATFULL
AN     2002:165194  USPATFULL
TI     Nucleic acids, proteins, and antibodies
IN     Rosen, Craig A., Laytonsville, MD, UNITED STATES
       Ruben, Steven M., Olney, MD, UNITED STATES
       Barash, Steven C., Rockville, MD, UNITED STATES
PI     US 2002086823      A1   20020704
AI     US 2001-764889     A1   20010117 (9)
PRAI   US 2000-179065P     20000131 (60)
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DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 17471
AB The present invention relates to novel respiratory system related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "respiratory system antigens," and the use of such respiratory system antigens for detecting disorders of the respiratory system, particularly the presence of cancer of respiratory system tissues and cancer metastases. More specifically, isolated respiratory system associated nucleic acid molecules are provided encoding novel respiratory system associated polypeptides. Novel respiratory system polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human respiratory system associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the respiratory system, including cancer of respiratory system tissues, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

L21 ANSWER 3 OF 44 USPATFULL
AN 2002:165193 USPATFULL
TI Nucleic acids, proteins, and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Barash, Steven C., Rockville, MD, UNITED STATES
PI US 2002086822 A1 20020704
AI US 2001-764886 A1 20010117 (9)
PRAI US 2000-179065P 20000131 (60)
US 2000-180628P 20000204 (60)
US 2000-214886P 20000628 (60)
US 2000-217487P 20000711 (60)
US 2000-225758P 20000814 (60)
US 2000-220963P 20000726 (60)
US 2000-217496P 20000711 (60)
US 2000-225447P 20000814 (60)
US 2000-218290P 20000714 (60)
US 2000-225757P 20000814 (60)
US 2000-226868P 20000822 (60)
US 2000-216647P 20000707 (60)
US 2000-225267P 20000814 (60)
US 2000-216880P 20000707 (60)
US 2000-225270P 20000814 (60)
US 2000-251869P 20001208 (60)
US 2000-235834P 20000927 (60)
US 2000-234274P 20000921 (60)
US 2000-234223P 20000921 (60)
US 2000-228924P 20000830 (60)
US 2000-224518P 20000814 (60)
US 2000-236369P 20000929 (60)
US 2000-224519P 20000814 (60)
US 2000-220964P 20000726 (60)
US 2000-241809P 20001020 (60)

US 2000-249299P 20001117 (60)
US 2000-236327P 20000929 (60)
US 2000-241785P 20001020 (60)
US 2000-244617P 20001101 (60)
US 2000-225268P 20000814 (60)
US 2000-236368P 20000929 (60)
US 2000-251856P 20001208 (60)
US 2000-251868P 20001208 (60)
US 2000-229344P 20000901 (60)
US 2000-234997P 20000925 (60)
US 2000-229343P 20000901 (60)
US 2000-229345P 20000901 (60)
US 2000-229287P 20000901 (60)
US 2000-229513P 20000905 (60)
US 2000-231413P 20000908 (60)
US 2000-229509P 20000905 (60)
US 2000-236367P 20000929 (60)
US 2000-237039P 20001002 (60)
US 2000-237038P 20001002 (60)
US 2000-236370P 20000929 (60)
US 2000-236802P 20001002 (60)
US 2000-237037P 20001002 (60)
US 2000-237040P 20001002 (60)
US 2000-240960P 20001020 (60)
US 2000-239935P 20001013 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 20931
AB The present invention relates to novel proteins. More specifically, isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

L21 ANSWER 4 OF 44 USPATFULL
AN 2002:165192 USPATFULL
TI Nucleic acids, proteins, and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Barash, Steven C., Rockville, MD, UNITED STATES
PI US 2002086821 A1 20020704
AI US 2001-764881 A1 20010117 (9)
PRAI US 2000-179065P 20000131 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 27531

AB The present invention relates to novel respiratory system related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "respiratory system antigens," and the use of such respiratory system antigens for detecting disorders of the respiratory system, particularly the presence of cancer of respiratory system tissues and cancer metastases. More specifically, isolated respiratory system associated nucleic acid molecules are provided encoding novel respiratory system associated polypeptides. Novel respiratory system polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human respiratory system associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the respiratory system, including cancer of respiratory system tissues, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

L21 ANSWER 5 OF 44 USPATFULL
AN 2002:165191 USPATFULL
TI Nucleic acids, proteins, and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Barash, Steven C., Rockville, MD, UNITED STATES
PI US 2002086820 A1 20020704
AI US 2001-764862 A1 20010117 (9)
PRAI US 2000-179065P 20000131 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 17727
AB The present invention relates to novel respiratory system related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "respiratory system antigens," and the use of such respiratory system antigens for detecting disorders of the respiratory system, particularly the presence of cancer of respiratory system tissues and cancer metastases. More specifically, isolated respiratory system associated nucleic acid molecules are provided encoding novel respiratory system associated polypeptides. Novel respiratory system polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human respiratory system associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the respiratory system, including cancer of respiratory system tissues, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

L21 ANSWER 6 OF 44 USPATFULL
AN 2002:165182 USPATFULL

TI Nucleic acids, proteins, and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Barash, Steven C., Rockville, MD, UNITED STATES
PI US 2002086811 A1 20020704
AI US 2001-764861 A1 20010117 (9)
PRAI US 2000-179065P 20000131 (60)
US 2000-180628P 20000204 (60)
US 2000-214886P 20000628 (60)
US 2000-217487P 20000711 (60)
US 2000-225758P 20000814 (60)
US 2000-220963P 20000726 (60)
US 2000-217496P 20000711 (60)
US 2000-225447P 20000814 (60)
US 2000-218290P 20000714 (60)
US 2000-225757P 20000814 (60)
US 2000-226868P 20000822 (60)
US 2000-216647P 20000707 (60)
US 2000-225267P 20000814 (60)
US 2000-216880P 20000707 (60)
US 2000-225270P 20000814 (60)
US 2000-251869P 20001208 (60)
US 2000-235834P 20000927 (60)
US 2000-234274P 20000921 (60)
US 2000-234223P 20000921 (60)
US 2000-228924P 20000830 (60)
US 2000-224518P 20000814 (60)
US 2000-236369P 20000929 (60)
US 2000-224519P 20000814 (60)
US 2000-220964P 20000726 (60)
US 2000-241809P 20001020 (60)
US 2000-249299P 20001117 (60)
US 2000-236327P 20000929 (60)
US 2000-241785P 20001020 (60)
US 2000-244617P 20001101 (60)
US 2000-225268P 20000814 (60)
US 2000-236368P 20000929 (60)
US 2000-251856P 20001208 (60)
US 2000-251868P 20001208 (60)
US 2000-229344P 20000901 (60)
US 2000-234997P 20000925 (60)
US 2000-229343P 20000901 (60)
US 2000-229345P 20000901 (60)
US 2000-229287P 20000901 (60)
US 2000-229513P 20000905 (60)
US 2000-231413P 20000908 (60)
US 2000-229509P 20000905 (60)
US 2000-236367P 20000929 (60)
US 2000-237039P 20001002 (60)
US 2000-237038P 20001002 (60)
US 2000-236370P 20000929 (60)
US 2000-236802P 20001002 (60)
US 2000-237037P 20001002 (60)
US 2000-237040P 20001002 (60)
US 2000-240960P 20001020 (60)
US 2000-239935P 20001013 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN No Drawings

LN.CNT 22023

AB The present invention relates to novel proteins. More specifically, isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

L21 ANSWER 7 OF 44 USPATFULL

AN 2002:164735 USPATFULL

TI Nucleic acids, proteins, and **antibodies**

IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Barash, Steven C., Rockville, MD, UNITED STATES

PI US 2002086353 A1 20020704

AI US 2001-764856 A1 20010117 (9)

PRAI US 2000-179065P 20000131 (60)
US 2000-180628P 20000204 (60)
US 2000-214886P 20000628 (60)
US 2000-217487P 20000711 (60)
US 2000-225758P 20000814 (60)
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US 2000-216647P 20000707 (60)
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US 2000-249299P 20001117 (60)
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US 2000-229345P 20000901 (60)
US 2000-229287P 20000901 (60)
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US 2000-231413P 20000908 (60)
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US 2000-236370P 20000929 (60)
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US 2000-237037P 20001002 (60)
US 2000-237040P 20001002 (60)
US 2000-240960P 20001020 (60)
US 2000-239935P 20001013 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 23314
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel proteins. More specifically, isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

L21 ANSWER 8 OF 44 USPATFULL
AN 2002:164712 USPATFULL
TI Nucleic acids, proteins, and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Barash, Steven C., Rockville, MD, UNITED STATES
PI US 2002086330 A1 20020704
AI US 2001-764893 A1 20010117 (9)
PRAI US 2000-179065P 20000131 (60)
US 2000-180628P 20000204 (60)
US 2000-214886P 20000628 (60)
US 2000-217487P 20000711 (60)
US 2000-225758P 20000814 (60)
US 2000-220963P 20000726 (60)
US 2000-217496P 20000711 (60)
US 2000-225447P 20000814 (60)
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US 2000-225757P 20000814 (60)
US 2000-226868P 20000822 (60)
US 2000-216647P 20000707 (60)
US 2000-225267P 20000814 (60)
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US 2000-236369P 20000929 (60)
US 2000-224519P 20000814 (60)
US 2000-220964P 20000726 (60)
US 2000-241809P 20001020 (60)
US 2000-249299P 20001117 (60)
US 2000-236327P 20000929 (60)
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US 2000-229513P 20000905 (60)
US 2000-231413P 20000908 (60)
US 2000-229509P 20000905 (60)
US 2000-236367P 20000929 (60)
US 2000-237039P 20001002 (60)
US 2000-237038P 20001002 (60)
US 2000-236370P 20000929 (60)
US 2000-236802P 20001002 (60)
US 2000-237037P 20001002 (60)
US 2000-237040P 20001002 (60)
US 2000-240960P 20001020 (60)
US 2000-239935P 20001013 (60)

DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 25862
AB The present invention relates to novel proteins. More specifically, isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

The present invention relates to novel proteins. More specifically, isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production

and function of the polypeptides of the present invention.

L21 ANSWER 9 OF 44 USPATFULL
AN 2002:157060 USPATFULL
TI Nucleic acids, proteins and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PI US 2002081659 A1 20020627
AI US 2001-925297 A1 20010810 (9)
RLI Continuation-in-part of Ser. No. WO 2000-US5989, filed on 8 Mar 2000, UNKNOWN
PRAI US 1999-124270P 19990312 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 20326
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel pancreatic related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "pancreatic antigens," and **antibodies** that immunospecifically bind these polypeptides, and the use of such pancreatic polynucleotides, antigens, and **antibodies** for detecting, treating, preventing and/or prognosing disorders of the pancreas, including, but not limited to, the presence of pancreatic cancer and pancreatic cancer metastases. More specifically, isolated pancreatic nucleic acid molecules are provided encoding novel pancreatic polypeptides. Novel pancreatic polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human pancreatic polynucleotides, polypeptides, and/or **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the pancreas, including pancreatic cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

L21 ANSWER 10 OF 44 USPATFULL
AN 2002:149114 USPATFULL
TI Nucleic acids, proteins, and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Barash, Steven C., Rockville, MD, UNITED STATES
PI US 2002077270 A1 20020620
AI US 2001-764848 A1 20010117 (9)
PRAI US 2000-179065P 20000131 (60)
US 2000-180628P 20000204 (60)
US 2000-214886P 20000628 (60)
US 2000-217487P 20000711 (60)
US 2000-225758P 20000814 (60)
US 2000-220963P 20000726 (60)
US 2000-217496P 20000711 (60)
US 2000-225447P 20000814 (60)
US 2000-218290P 20000714 (60)
US 2000-225757P 20000814 (60)
US 2000-226868P 20000822 (60)

US 2000-216647P 20000707 (60)
US 2000-225267P 20000814 (60)
US 2000-216880P 20000707 (60)
US 2000-225270P 20000814 (60)
US 2000-251869P 20001208 (60)
US 2000-235834P 20000927 (60)
US 2000-234274P 20000921 (60)
US 2000-234223P 20000921 (60)
US 2000-228924P 20000830 (60)
US 2000-224518P 20000814 (60)
US 2000-236369P 20000929 (60)
US 2000-224519P 20000814 (60)
US 2000-220964P 20000726 (60)
US 2000-241809P 20001020 (60)
US 2000-249299P 20001117 (60)
US 2000-236327P 20000929 (60)
US 2000-241785P 20001020 (60)
US 2000-244617P 20001101 (60)
US 2000-225268P 20000814 (60)
US 2000-236368P 20000929 (60)
US 2000-251856P 20001208 (60)
US 2000-251868P 20001208 (60)
US 2000-229344P 20000901 (60)
US 2000-234997P 20000925 (60)
US 2000-229343P 20000901 (60)
US 2000-229345P 20000901 (60)
US 2000-229287P 20000901 (60)
US 2000-229513P 20000905 (60)
US 2000-231413P 20000908 (60)
US 2000-229509P 20000905 (60)
US 2000-236367P 20000929 (60)
US 2000-237039P 20001002 (60)
US 2000-237038P 20001002 (60)
US 2000-236370P 20000929 (60)
US 2000-236802P 20001002 (60)
US 2000-237037P 20001002 (60)
US 2000-237040P 20001002 (60)
US 2000-240960P 20001020 (60)
US 2000-239935P 20001013 (60)

DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 20057
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel proteins. More specifically, isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

L21 ANSWER 11 OF 44 USPATFULL

AN 2002:148255 USPATFULL
TI Membrane-bound cytokine compositions and methods of modulating an immune response using same
IN Hoo, William Soo, Carlsbad, CA, UNITED STATES
PI US 2002076392 A1 20020620
AI US 2001-847185 A1 20010501 (9)
RLI Continuation of Ser. No. US 1998-201931, filed on 1 Dec 1998, PENDING
Continuation of Ser. No. US 1997-902516, filed on 29 Jul 1997, GRANTED, Pat. No. US 5891432
DT Utility
FS APPLICATION
LREP CAMPBELL & FLORES LLP, 4370 LA JOLLA VILLAGE DRIVE, 7TH FLOOR, SAN DIEGO, CA, 92122
CLMN Number of Claims: 46
ECL Exemplary Claim: 1
DRWN 4 Drawing Page(s)
LN.CNT 1978
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule operatively fused to a heterologous membrane attachment domain. Non-**antibody** immunomodulatory molecules useful in the invention include immunostimulatory and immunosuppressive molecules such as cytokines. In one embodiment, the invention provides a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule operatively fused to a heterologous membrane attachment domain and, additionally, a disease-associated antigen or immunogenic **epitope** thereof. Further provided by the invention are methods of modulating an immune response against a disease-associated antigen by administering to an individual a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule operatively fused to a heterologous membrane attachment domain.

L21 ANSWER 12 OF 44 USPATFULL
AN 2002:133211 USPATFULL
TI Cytokine antagonists
IN Debets, Johannes Eduard Maria Antonius, Rhoon, NETHERLANDS
Abrams, John S., Los Altos, CA, UNITED STATES
Kastelein, Robert A., Redwood City, CA, UNITED STATES
O'Garra, Anne, Palo Alto, CA, UNITED STATES
PI US 2002068060 A1 20020606
AI US 2001-834295 A1 20010412 (9)
PRAI US 2000-196754P 20000412 (60)
DT Utility
FS APPLICATION
LREP SCHERING-PLOUGH CORPORATION, PATENT DEPARTMENT (K-6-1, 1990), 2000 GALLOPING HILL ROAD, KENILWORTH, NJ, 07033-0530
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 862
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Antagonists of a cytokine signaling system have been found which exhibit favorable properties. In particular, **antibody** antagonists raised against the receptor are effective in blocking various signaling processes.

L21 ANSWER 13 OF 44 USPATFULL
AN 2002:119538 USPATFULL
TI Nucleic acids, proteins, and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES

Ruben, Steven M., Olney, MD, UNITED STATES
Barash, Steven C., Rockville, MD, UNITED STATES
PI US 2002061521 A1 20020523
AI US 2001-764869 A1 20010117 (9)
PRAI US 2000-179065P 20000131 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 27967
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel cardiovascular system related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "cardiovascular system antigens," and the use of such cardiovascular system antigens for detecting disorders of the cardiovascular system, particularly the presence of cancer of cardiovascular system tissues and cancer metastases. More specifically, isolated cardiovascular system associated nucleic acid molecules are provided encoding novel cardiovascular system associated polypeptides. Novel cardiovascular system polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human cardiovascular system associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the cardiovascular system, including cancer of cardiovascular system tissues, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

L21 ANSWER 14 OF 44 USPATFULL
AN 2002:106416 USPATFULL
TI Nucleic acids, proteins and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PI US 2002055627 A1 20020509
AI US 2001-925299 A1 20010810 (9)
RLI Continuation of Ser. No. WO 2000-US5883, filed on 8 Mar 2000, UNKNOWN
PRAI US 1999-124270P 19990312 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 20658
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel colorectal cancer related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "colorectal cancer antigens," and **antibodies** that immunospecifically bind these polypeptides, and the use of such colorectal cancer polynucleotides, antigens, and **antibodies** for detecting, treating, preventing and/or prognosing disorders of the colon and/or rectum, including, but not limited to, the presence of colorectal cancer and colorectal cancer metastases. More specifically, isolated colorectal cancer nucleic acid molecules are provided encoding novel colorectal cancer polypeptides. Novel colorectal

cancer polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human colorectal cancer polynucleotides, polypeptides, and/or **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the colon and/or rectum, including colorectal cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

L21 ANSWER 15 OF 44 USPATFULL
AN 2002:99407 USPATFULL
TI Nucleic acids, proteins and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PI US 2002052308 A1 20020502
AI US 2001-925301 A1 20010810 (9)
RLI Continuation of Ser. No. WO 2000-US5882, filed on 8 Mar 2000, UNKNOWN
PRAI US 1999-124270P 19990312 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 30577
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to newly identified tissue specific cancer associated polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "cancer antigens," and to the complete gene sequences associated therewith and to the expression products thereof, as well as the use of such tissue specific cancer antigens for detection, prevention and treatment of tissue specific disorders, particularly the presense of cancer. This invention relates to the cancer antigens as well as vectors, host cells, **antibodies** directed to cancer antigens and recombinant and synthetic methods for producing the same. Also provided are diagnostic methods for diagnosing and treating, preventing and/or prognosing tissue specific disorders, including cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of cancer antigens of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and/or function of the polypeptides of the present invention.

L21 ANSWER 16 OF 44 USPATFULL
AN 2002:85190 USPATFULL
TI Nucleic acids, proteins, and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Rubin, Steven M., Olney, MD, UNITED STATES
Barash, Steven C., Rockville, MD, UNITED STATES
PI US 2002045230 A1 20020418
AI US 2001-908711 A1 20010720 (9)
RLI Continuation-in-part of Ser. No. WO 2001-US1360, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764867, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1344, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764892, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1345, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser.

No. US 2001-764888, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1329, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764905, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764891, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1339, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764869, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1340, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764874, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1334, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764898, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1320, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764853, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764902, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1239, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764870, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1348, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764882, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1347, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764896, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1307, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764864, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1341, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764856, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1336, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764868, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1312, filed on 17 Jan 2001, UNKNOWN

PRAI	US 2000-179065P	20000131	(60)
	US 2000-180628P	20000204	(60)
	US 2000-251868P	20001208	(60)
	US 2000-232398P	20000914	(60)
	US 2000-249300P	20001117	(60)
	US 2000-251990P	20001208	(60)
	US 2000-250160P	20001201	(60)
	US 2000-209467P	20000607	(60)
	US 2000-179065P	20000131	(60)
	US 2000-180628P	20000204	(60)
	US 2000-214886P	20000628	(60)
	US 2000-217487P	20000711	(60)
	US 2000-225758P	20000814	(60)
	US 2000-220963P	20000726	(60)
	US 2000-217496P	20000711	(60)
	US 2000-225447P	20000814	(60)
	US 2000-218290P	20000714	(60)
	US 2000-225757P	20000814	(60)
	US 2000-226868P	20000822	(60)
	US 2000-216647P	20000707	(60)
	US 2000-225267P	20000814	(60)
	US 2000-216880P	20000707	(60)
	US 2000-225270P	20000814	(60)
	US 2000-251869P	20001208	(60)
	US 2000-235834P	20000927	(60)
	US 2000-234274P	20000921	(60)
	US 2000-234223P	20000921	(60)
	US 2000-228924P	20000830	(60)
	US 2000-224518P	20000814	(60)
	US 2000-236369P	20000929	(60)
	US 2000-224519P	20000814	(60)
	US 2000-220964P	20000726	(60)

US 2000-241809P 20001020 (60)
US 2000-249299P 20001117 (60)
US 2000-236327P 20000929 (60)
US 2000-241785P 20001020 (60)
US 2000-244617P 20001101 (60)
US 2000-225268P 20000814 (60)
US 2000-236368P 20000929 (60)
US 2000-251856P 20001208 (60)
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US 2000-229343P 20000901 (60)
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US 2000-231413P 20000908 (60)
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US 2000-241787P 20001020 (60)
US 2000-246474P 20001108 (60)
US 2000-246532P 20001108 (60)
US 2000-249216P 20001117 (60)
US 2000-249210P 20001117 (60)
US 2000-226681P 20000822 (60)
US 2000-225759P 20000814 (60)
US 2000-225213P 20000814 (60)
US 2000-227182P 20000822 (60)
US 2000-225214P 20000814 (60)
US 2000-235836P 20000927 (60)
US 2000-230438P 20000906 (60)
US 2000-215135P 20000630 (60)
US 2000-225266P 20000814 (60)
US 2000-249218P 20001117 (60)
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US 2000-249207P 20001117 (60)
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US 2000-249211P 20001117 (60)
US 2000-249215P 20001117 (60)
US 2000-249264P 20001117 (60)
US 2000-249214P 20001117 (60)
US 2000-249297P 20001117 (60)
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US 2000-231242P 20000908 (60)
US 2000-232081P 20000908 (60)
US 2000-232080P 20000908 (60)
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US 2000-231244P 20000908 (60)
US 2000-233064P 20000914 (60)
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US 2000-232399P 20000914 (60)
US 2000-232401P 20000914 (60)
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US 2000-241826P 20001020 (60)
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US 2000-234998P 20000925 (60)
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US 2000-246525P 20001108 (60)
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US 2000-251988P 20001205 (60)
US 2000-251030P 20001205 (60)
US 2000-251479P 20001206 (60)
US 2000-256719P 20001205 (60)
US 2000-250160P 20001201 (60)
US 2000-251989P 20001208 (60)
US 2000-250391P 20001201 (60)
US 2000-254097P 20001211 (60)
US 2000-231968P 20000912 (60)
US 2000-226279P 20000818 (60)
US 2000-186350P 20000302 (60)
US 2000-184664P 20000224 (60)
US 2000-189874P 20000316 (60)
US 2000-198123P 20000418 (60)
US 2000-227009P 20000823 (60)
US 2000-235484P 20000926 (60)
US 2000-190076P 20000317 (60)
US 2000-209467P 20000607 (60)
US 2000-205515P 20000519 (60)
US 2001-259678P 20010105 (60)
US 2000-179065P 20000131 (60)
US 2000-180628P 20000204 (60)
US 2000-214886P 20000628 (60)
US 2000-217487P 20000711 (60)
US 2000-225758P 20000814 (60)
US 2000-220963P 20000726 (60)
US 2000-217496P 20000711 (60)
US 2000-225447P 20000814 (60)
US 2000-218290P 20000714 (60)
US 2000-225757P 20000814 (60)
US 2000-226868P 20000822 (60)
US 2000-216647P 20000707 (60)

US	2000-225267P	20000814 (60)
US	2000-216880P	20000707 (60)
US	2000-225270P	20000814 (60)
US	2000-251869P	20001208 (60)
US	2000-235834P	20000927 (60)
US	2000-234274P	20000921 (60)
US	2000-234223P	20000921 (60)
US	2000-228924P	20000830 (60)
US	2000-224518P	20000814 (60)
US	2000-236369P	20000929 (60)
US	2000-224519P	20000814 (60)
US	2000-220964P	20000726 (60)
US	2000-241809P	20001020 (60)
US	2000-249299P	20001117 (60)
US	2000-236327P	20000929 (60)
US	2000-241785P	20001020 (60)
US	2000-244617P	20001101 (60)
US	2000-225268P	20000814 (60)
US	2000-236368P	20000929 (60)
US	2000-251856P	20001208 (60)
US	2000-251868P	20001208 (60)
US	2000-229344P	20000901 (60)
US	2000-234997P	20000925 (60)
US	2000-229343P	20000901 (60)
US	2000-229345P	20000901 (60)
US	2000-229287P	20000901 (60)
US	2000-229513P	20000905 (60)
US	2000-231413P	20000908 (60)
US	2000-229509P	20000905 (60)
US	2000-236367P	20000929 (60)
US	2000-237039P	20001002 (60)
US	2000-237038P	20001002 (60)
US	2000-236370P	20000929 (60)
US	2000-236802P	20001002 (60)
US	2000-237037P	20001002 (60)
US	2000-237040P	20001002 (60)
US	2000-240960P	20001020 (60)
US	2000-239935P	20001013 (60)
US	2000-239937P	20001013 (60)
US	2000-241787P	20001020 (60)
US	2000-246474P	20001108 (60)
US	2000-246532P	20001108 (60)
US	2000-249216P	20001117 (60)
US	2000-249210P	20001117 (60)
US	2000-226681P	20000822 (60)
US	2000-225759P	20000814 (60)
US	2000-225213P	20000814 (60)
US	2000-227182P	20000822 (60)
US	2000-225214P	20000814 (60)
US	2000-235836P	20000927 (60)
US	2000-230438P	20000906 (60)
US	2000-215135P	20000630 (60)
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US 2001-259678P 20010105 (60)

DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 24462
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel ovarian related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "ovarian antigens," and the use of such ovarian antigens for detecting disorders of the ovaries and/or breast,

particularly the presence of ovarian and/or breast cancer and ovarian and/or breast cancer metastases. More specifically, isolated ovarian associated nucleic acid molecules are provided encoding novel ovarian associated polypeptides. Novel ovarian polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human ovarian associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the ovaries and/or breast, including ovarian and/or breast cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

L21 ANSWER 17 OF 44 USPATFULL
AN 2002:84902 USPATFULL
TI Nucleic acids, proteins and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PI US 2002044941 A1 20020418
AI US 2001-925302 A1 20010810 (9)
RLI Continuation-in-part of Ser. No. WO 2000-US5918, filed on 8 Mar 2000, UNKNOWN
PRAI US 1999-124270P 19990312 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 21121
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel lung cancer related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "lung cancer antigens," and **antibodies** that immunospecifically bind these polypeptides, and the use of such lung cancer polynucleotides, antigens, and **antibodies** for detecting, treating, preventing and/or prognosing disorders of the lung, including, but not limited to, the presence of lung cancer and lung cancer metastases. More specifically, isolated lung cancer nucleic acid molecules are provided encoding novel lung cancer polypeptides. Novel lung cancer polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human lung cancer polynucleotides, polypeptides, and/or **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the lung, including lung cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

L21 ANSWER 18 OF 44 USPATFULL
AN 2002:78729 USPATFULL
TI Nucleic acids, proteins, and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES

Barash, Steven C., Rockville, MD, UNITED STATES
PI US 2002042386 A1 20020411
AI US 2001-764870 A1 20010117 (9)
PRAI US 2000-179065P 20000131 (60)
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US 2000-239935P 20001013 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 23133
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel proteins. More specifically,

isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

L21 ANSWER 19 OF 44 USPATFULL
AN 2002:78442 USPATFULL
TI Nucleic acids, proteins, and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Barash, Steven C., Rockville, MD, UNITED STATES
PI US 2002042096 A1 20020411
AI US 2001-764887 A1 20010117 (9)
PRAI US 2000-179065P 20000131 (60)
US 2000-180628P 20000204 (60)
US 2000-214886P 20000628 (60)
US 2000-217487P 20000711 (60)
US 2000-225758P 20000814 (60)
US 2000-220963P 20000726 (60)
US 2000-217496P 20000711 (60)
US 2000-225447P 20000814 (60)
US 2000-218290P 20000714 (60)
US 2000-225757P 20000814 (60)
US 2000-226868P 20000822 (60)
US 2000-216647P 20000707 (60)
US 2000-225267P 20000814 (60)
US 2000-216880P 20000707 (60)
US 2000-225270P 20000814 (60)
US 2000-251869P 20001208 (60)
US 2000-235834P 20000927 (60)
US 2000-234274P 20000921 (60)
US 2000-234223P 20000921 (60)
US 2000-228924P 20000830 (60)
US 2000-224518P 20000814 (60)
US 2000-236369P 20000929 (60)
US 2000-224519P 20000814 (60)
US 2000-220964P 20000726 (60)
US 2000-241809P 20001020 (60)
US 2000-249299P 20001117 (60)
US 2000-236327P 20000929 (60)
US 2000-241785P 20001020 (60)
US 2000-244617P 20001101 (60)
US 2000-225268P 20000814 (60)
US 2000-236368P 20000929 (60)
US 2000-251856P 20001208 (60)
US 2000-251868P 20001208 (60)
US 2000-229344P 20000901 (60)
US 2000-234997P 20000925 (60)
US 2000-229343P 20000901 (60)
US 2000-229345P 20000901 (60)
US 2000-229287P 20000901 (60)
US 2000-229513P 20000905 (60)
US 2000-231413P 20000908 (60)
US 2000-229509P 20000905 (60)

US 2000-236367P 20000929 (60)
US 2000-237039P 20001002 (60)
US 2000-237038P 20001002 (60)
US 2000-236370P 20000929 (60)
US 2000-236802P 20001002 (60)
US 2000-237037P 20001002 (60)
US 2000-237040P 20001002 (60)
US 2000-240960P 20001020 (60)
US 2000-239935P 20001013 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 19583
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel liver related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "liver antigens," and the use of such liver antigens for detecting disorders of the liver, particularly the presence of cancer of liver and cancer metastases. More specifically, isolated liver associated nucleic acid molecules are provided encoding novel liver associated polypeptides. Novel liver polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human liver associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the liver, including cancer of liver tissues, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

L21 ANSWER 20 OF 44 USPATFULL
AN 2002:72627 USPATFULL
TI Nucleic, acids, proteins, and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PI US 2002039764 A1 20020404
AI US 2001-925298 A1 20010810 (9)
RLI Continuation-in-part of Ser. No. WO 2000-US5881, filed on 8 Mar 2000, UNKNOWN
PRAI US 1999-124270P 19990312 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 20087
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel ovarian cancer and/or breast cancer related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "ovarian and/or breast antigens," and **antibodies** that immunospecifically bind these polypeptides, and the use of such ovarian and/or breast polynucleotides, antigens, and **antibodies** for detecting, treating, preventing and/or prognosing disorders of the reproductive system, particularly disorders of the ovaries and/or breast, including,

but not limited to, the presence of ovarian and/or breast cancer and ovarian and/or breast cancer metastases. More specifically, isolated ovarian and/or breast nucleic acid molecules are provided encoding novel ovarian and/or breast polypeptides. Novel ovarian and/or breast polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human ovarian and/or breast polynucleotides, polypeptides, and/or **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the ovaries and/or breast, including ovarian and/or breast cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

L21 ANSWER 21 OF 44 USPATFULL
AN 2002:72462 USPATFULL
TI Methods of diagnosing and treating small intestinal bacterial overgrowth (SIBO) and SIBO-related conditions
IN Lin, Henry C., Manhattan Beach, CA, UNITED STATES
Pimentel, Mark, Los Angeles, CA, UNITED STATES
PI US 2002039599 A1 20020404
AI US 2001-837797 A1 20010417 (9)
RLI Continuation-in-part of Ser. No. US 1999-374142, filed on 11 Aug 1999, PENDING Continuation-in-part of Ser. No. US 2000-546119, filed on 10 Apr 2000, PENDING Continuation-in-part of Ser. No. US 1999-420046, filed on 18 Oct 1999, PENDING Continuation-in-part of Ser. No. US 1999-359583, filed on 22 Jul 1999, ABANDONED Continuation of Ser. No. US 1997-832307, filed on 3 Apr 1997, GRANTED, Pat. No. US 5977175 Continuation of Ser. No. US 1995-442843, filed on 17 May 1995, ABANDONED
DT Utility
FS APPLICATION
LREP SIDLEY & AUSTIN, 555 West Fifth Street, Los Angeles, CA, 90071-2909
CLMN Number of Claims: 45
ECL Exemplary Claim: 1
DRWN 13 Drawing Page(s)
LN.CNT 4226
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a method of treating small intestinal bacterial overgrowth (SIBO) or a SIBO-caused condition in a human subject. SIBO-caused conditions include irritable bowel syndrome, fibromyalgia, chronic pelvic pain syndrome, chronic fatigue syndrome, depression, impaired mentation, impaired memory, halitosis, tinnitus, sugar craving, autism, attention deficit/hyperactivity disorder, drug sensitivity, an autoimmune disease, and Crohn's disease. Also disclosed are a method of screening for the abnormally likely presence of SIBO in a human subject and a method of detecting SIBO in a human subject. A method of determining the relative severity of SIBO or a SIBO-caused condition in a human subject, in whom small intestinal bacterial overgrowth (SIBO) has been detected, is also disclosed.

L21 ANSWER 22 OF 44 USPATFULL
AN 2002:67349 USPATFULL
TI Coupling of peripheral tolerance to endogenous IL-10 promotes effective modulation of T cells and ameliorates autoimmune disease
IN Zaghouani, Habib, Columbia, MO, UNITED STATES
PI US 2002038002 A1 20020328
AI US 2001-873901 A1 20010604 (9)
PRAI US 2000-209527P 20000605 (60)

DT Utility
FS APPLICATION
LREP KNOBBE MARTENS OLSON & BEAR LLP, 620 NEWPORT CENTER DRIVE, SIXTEENTH FLOOR, NEWPORT BEACH, CA, 92660
CLMN Number of Claims: 65
ECL Exemplary Claim: 1
DRWN 45 Drawing Page(s)
LN.CNT 4140
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Immunomodulating agents comprising at least one Fc receptor ligand and at least one immunosuppressive factor are provided as are methods for their manufacture and use. The immunomodulating agents may be in the form of polypeptides or chimeric **antibodies** and preferably incorporate an immunosuppressive factor comprising a T cell receptor agonist or **antagonist**. The compounds and compositions of the invention may be used to selectively suppress the immune system to treat symptoms associated with immune disorders such as allergies, transplanted tissue rejection and autoimmune disorders including autoimmune diabetes, **rheumatoid arthritis** and multiple sclerosis.

L21 ANSWER 23 OF 44 USPATFULL
AN 2002:48631 USPATFULL
TI THERAPEUTIC COMPOUNDS FOR INHIBITING INTERLEUKIN-12 SIGNALING AND METHODS FOR USING SAME
IN KLEIN, J. PETER, VASHON, WA, UNITED STATES
KLAUS, STEPHEN J., SEATTLE, WA, UNITED STATES
KUMAR, ANIL M., MERCER ISLAND, WA, UNITED STATES
GONG, BAOQING, SHORELINE, WA, UNITED STATES
PI US 2002028823 A1 20020307
AI US 1999-288556 A1 19990409 (9)
RLI Continuation-in-part of Ser. No. US 1998-8020, filed on 16 Jan 1998, ABANDONED
DT Utility
FS APPLICATION
LREP MCDERMOTT WILL & EMERY, 600 13TH STREET, N.W., WASHINGTON, DC, 20005-3096
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 1 Drawing Page(s)
LN.CNT 4381
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel heterocyclic compounds having a six membered ring structure fused to a five membered ring structure are found to be useful for the treatment and prevention of symptoms or manifestations associated with disorders affected by Interleukin-12 ("**IL-12**") intracellular signaling, such as, for example, Th1 cell-mediated disorders. The therapeutic compounds, pharmaceutically acceptable derivatives (e.g., resolved enantiomers, diastereomers, tautomers, salts and solvates thereof) or prodrugs thereof, have the following general formula: ##STR1##

Each X, Y and Z are independently selected from a member of the group consisting of C(R.sub.3), N, N(R.sub.3) and S. Each R.sub.1, R.sub.2 and R.sub.3 is substituted or unsubstituted and is independently selected from a member of the group consisting of hydrogen, halo, oxo, C.sub.(1-20)alkyl, C.sub.(1-20)hydroxyalkyl, C.sub.(1-20)thioalkyl, C.sub.(1-20)alkylamino, C.sub.(1-20)alkylaminoalkyl, C.sub.(1-20)aminoalkyl, C.sub.(1-20)aminoalkoxyalkenyl, C.sub.(1-20)aminoalkoxyalkynyl, C.sub.(1-20)diaminoalkyl, C.sub.(1-20)triaminoalkyl, C.sub.(1-20)tetraaminoalkyl, C.sub.(5-15)aminotrialkoxyamino, C.sub.(1-20)alkylamido,

C.sub.(1-20)alkylamidoalkyl, C.sub.(1-20)amidoalkyl, C.sub.(1-20)acetamidoalkyl, C.sub.(1-20)alkenyl, C.sub.(1-20)alkynyl, C.sub.(3-8)alkoxyl, C.sub.(1-11)alkoxyalkyl, and C.sub.(1-20)dialkoxyalkyl.

L21 ANSWER 24 OF 44 USPATFULL
AN 2002:42953 USPATFULL
TI Bispecific monoclonal **antibodies** to **IL-12** and IL-18
IN Leung, Stewart, El Cerrito, CA, UNITED STATES
Perez, H. Daniel, Kentfield, CA, UNITED STATES
Miyamoto, Neil, Corte Madera, CA, UNITED STATES
PA Schering AG, Berlin, GERMANY, FEDERAL REPUBLIC OF (U.S. corporation)
PI US 2002025317 A1 20020228
AI US 2001-907960 A1 20010719 (9)
PRAI US 2000-219448P 20000720 (60)
DT Utility
FS APPLICATION
LREP MILLEN, WHITE, ZELANO & BRANIGAN, P.C., 2200 CLARENDON BLVD., SUITE 1400, ARLINGTON, VA, 22201
CLMN Number of Claims: 30
ECL Exemplary Claim: 1
DRWN 7 Drawing Page(s)
LN.CNT 1451
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A bispecific monoclonal **antibody** is described which comprises two moieties, one of which comprises an antigen-binding region which is specific for either the IL-12R.beta.1 or the IL-12R.beta.2 subunit of an **IL-12** receptor, and the other of which comprises an antigen-binding region which is specific for either the IL-18R or the AcPL subunit of an IL-18 receptor.

L21 ANSWER 25 OF 44 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 2
AN 2001-244697 [25] WPIDS
DNC C2001-073427
TI Modulating responsiveness to a corticosteroid by administering a corticosteroid with an agent which antagonizes a target that regulates interferon-gamma production or an caspase family protease inhibitor, useful for treating asthma.
DC B04 B05 D16
IN BANERJEE, S; CARTER, A; GHAYUR, T; SEKUT, L; TRACEY, D E
PA (BADI) BASF AG
CYC 94
PI WO 2001019373 A2 20010322 (200125)* EN 152p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2000071276 A 20010417 (200140)
ADT WO 2001019373 A2 WO 2000-US24725 20000908; AU 2000071276 A AU 2000-71276 20000908
FDT AU 2000071276 A Based on WO 200119373
PRAI US 1999-398555 19990917
AB WO 200119373 A UPAB: 20010508
NOVELTY - A new method (M1) for modulating responsiveness to a corticosteroid in a subject comprises administering a corticosteroid with an agent (A1) which antagonizes a target that regulates production of interferon-gamma (IFN-gamma) or at least one agent (A2) that is an inhibitor of a caspase family protease.
DETAILED DESCRIPTION - A method (M1) for modulating responsiveness to

a corticosteroid in a subject, comprising selecting a subject in need of modulation of responsiveness to a corticosteroid and administering:

(a) an agent (A1) which antagonizes a target that regulates production of interferon-gamma (IFN-gamma) in the subject, the agent being administered at a dosage and by a route sufficient to inhibit production of IFN-gamma; or

(b) at least one agent (A2) that is an inhibitor of a caspase family protease; and

(c) a corticosteroid.

The responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.

An INDEPENDENT CLAIM is also given for a method (M2) for regulating the production of IFN-gamma in a subject, comprising administering a corticosteroid and an agent which antagonizes a target that regulates production of IFN-gamma such that production of IFN-gamma is modulated in the subject.

ACTIVITY - Immunosuppressive; antiinflammatory; dermatological; antibacterial; cytostatic; antiasthmatic; anticonvulsant; antidiabetic; antiarthritic; antirheumatic; neuroprotective; antiallergic; antiulcer; ophthalmological; antianemic.

Interleukin converting enzyme (ICE)-deficient and wild type mice first were sensitized with Propionibacterium acnes cell wall material (1 mg per mouse) to induce low grade inflammation and six days later were challenged with lipopolysaccharide (LPS) (1 microgram per mouse in 0.1 ml of saline intravenously). Thirty minutes after LPS administration, the mice were treated with the corticosteroid dexamethasone (4 mg/kg per mouse in 0.5 ml 95% saline/0.5% ethanol, intraperitoneally). Control mice were treated with vehicle alone. All mice were bled 90 minutes after LPS administration and the serum samples were analyzed for the presence of tumor necrosis alpha (TNF-alpha) by standard ELISA (Enzyme linked immunosorbant assay).

Wild type and ICE deficient mice treated with vehicle alone had similar levels of serum TNF-alpha. Treatment of wild type mice with dexamethasone did not significantly affect serum TNF-alpha levels, demonstrating their resistance to steroid treatment in this septic shock model. In contrast, treatment of the ICE deficient mice with dexamethasone suppressed serum TNF-alpha levels by 74% (p less than 0.002). These data indicate that inhibition of ICE activity reverses resistance to steroid treatment in a septic shock model.

MECHANISM OF ACTION - **IL-12 antagonist**; IL-18 **antagonist**; phosphodiesterase IV inhibitor; a beta-2 agonist; a STAT4 inhibitor; an anti-IL-1-alpha **antibody**; an anti-IL-1-beta **antibody**; an anti-tumor necrosis factor **antibody**; a natural killer cell **antagonist**; a T-cell **antagonist**; caspase family protease inhibitor; gene therapy.

USE - The method is useful for treating a subject suffering from an autoimmune disease or disorder, an acute (e.g. infectious meningitis) or chronic (e.g. systemic lupus erythematosus or psoriasis) inflammatory disorder, septic shock or sepsis, graft versus host disease or transplant rejection, complications associated with post-surgical stress, Still's disease, leukemia or an immuno-inflammatory disease or disorder. The immuno-inflammatory disease or disorder is asthma, adult respiratory distress syndrome, systemic lupus erythematosus, inflammatory bowel disease, Crohn's disease, ulcerative colitis, multiple sclerosis, insulin-dependent diabetes mellitus, autoimmune arthritis, **rheumatoid arthritis**, juvenile **rheumatoid arthritis**, psoriatic arthritis, inflammatory pulmonary syndrome, pemphigus vulgaris, idiopathic thrombocytopenic purpura, autoimmune meningitis, myasthenia gravis, autoimmune thyroiditis, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, Sjogren's Syndrome, keratoconjunctivitis sicca secondary to Sjogren's Syndrome, alopecia areata, allergic responses due to arthropod bite reactions, aphthous

ulcer, iritis, conjunctivitis, keratoconjunctivitis, cutaneous lupus erythematosus, scleroderma, vaginitis, proctitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, erythema nodosum leprosum, autoimmune uveitis, allergic encephalomyelitis, aplastic anemia, pure red cell anemia, idiopathic thrombocytopenia, polychondritis, Wegener's granulomatosis, chronic active hepatitis, Graves ophthalmopathy, primary biliary cirrhosis, uveitis posterior or interstitial lung fibrosis (claimed).

The method is useful for modulating corticosteroid responsiveness in a variety of clinical settings, for e.g. reversing steroid resistance, increasing steroid sensitivity, ameliorating a steroid rebound effect associated with administration of reduced dosages of the corticosteroid, or modulating corticosteroid activity, such that the corticosteroids can be tapered to zero (claimed).

Dwg.0/12

L21 ANSWER 26 OF 44 WPIDS (C) 2002 THOMSON DERWENT
AN 2002-075345 [10] WPIDS
DNC C2002-022528
TI Use of apoptotic bodies and/or apoptotic cells for treatment and prophylaxis of T cell-mediated and inflammatory disorders such as psoriasis, atherosclerosis, diabetes and scleroderma in mammalian patients.
DC B04 D16
IN BOLTON, A E; MANDEL, A; SAUDER, D
PA (VASO-N) VASOGEN IRELAND LTD; (BOLT-I) BOLTON A E; (MAND-I) MANDEL A; (SAUD-I) SAUDER D
CYC 94
PI WO 2001089536 A2 20011129 (200210)* EN 20p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
CA 2309518 A1 20011125 (200210) EN
AU 2001061986 A 20011203 (200221)
US 2002051771 A1 20020502 (200234)
ADT WO 2001089536 A2 WO 2001-CA758 20010525; CA 2309518 A1 CA 2000-2309518 20000525; AU 2001061986 A AU 2001-61986 20010525; US 2002051771 A1 US 2001-866488 20010525
FDT AU 2001061986 A Based on WO 200189536
PRAI CA 2000-2309518 20000525
AB WO 200189536 A UPAB: 20020213
NOVELTY - Use of apoptotic bodies and/or apoptotic cells for treatment and prophylaxis of T cell-mediated and inflammatory disorders, or in the preparation of a medicament for treatment and/or prophylaxis of T cell-mediated and inflammatory disorders in mammalian patients.

ACTIVITY - Antipsoriatic; antirheumatic; antiarthritic; immunosuppressive; neuroprotective; antiallergic; dermatological; antiinflammatory; antidiabetic; antiatherosclerotic; antiasthmatic; antimicrobial. The effectiveness of treatment on contact hypersensitivity (CHS), an example of a Th-1-cell inflammatory disorder was assessed on laboratory mice. To induce CHS, the abdominal skin of each mouse was painted with dinitrodifluorobenzene (DNFB). Apoptotic bodies were prepared from murine fibroblasts. The murine fibroblasts were treated with 50 nM sodium butyrate in RPMI (not defined) medium, at confluency for one day, and then the sodium butyrate medium was changed. To increase the number of apoptotic cells and bodies, the cells were additionally irradiated with ultra-violet (UV)-light. Supernatant containing floating cells was removed 24 hours following irradiation. Apoptotic bodies were quantitated. The pellet containing the apoptotic bodies was resuspended in phosphate

buffered saline (PBS). The cells to be stained for quantitation were resuspended in 1X binding buffer at a concentration of 1 multiply 106 cells/ml. Of the two groups of sensitized mice, the first, control group, received no treatment. The second, test group, was treated with an injection of suspended apoptotic bodies, 50 micro l volume containing at least 150000 bodies per injection of blood subjected to stressors. Treatments each involving intramuscular injection of 50 micro l of the respective liquid, started on the day of sensitization, and were repeated every day for a total of six days. On the same day as the last treatment, but after its administration, the animals were challenged with DNFB, by applying to the right ear to each animal 10 micro l of 0.2 % solution of DNFB in acetone and olive oil. To the left ear of each animal was applied the acetone/olive oil solvent without DNFB. Ear thickness was measured, 24 hours after challenge. The results were expressed as the thickness and difference in thickness of the right ears and the left ears of each animal, at 24 hours after challenge. The results showed a notable and significant reduction in ear thickness (inflammation) with the animals treated with the apoptotic cells and apoptotic bodies suspension, as compared with the untreated group, demonstrating a significant reduction in inflammation.

MECHANISM OF ACTION - Interleukin-10 (IL-10) agonist; tumor necrosis factor- gamma (TNF- gamma), IL-6 and **IL-12 antagonist**; vaccine.

USE - The method is useful for treatment and/or prophylaxis of T cell-mediated and inflammatory disorders such as psoriasis, **rheumatoid arthritis**, scleroderma, lupus, diabetes mellitus, organ rejection, miscarriage, multiple sclerosis, inflammatory bowel disease, atherosclerosis and graft versus host disease in mammalian patients (claimed), and also microbial infections, asthma, contact hypersensitivity and other inflammatory allergic reactions. The method is also applicable to preconditioning against ingestions of poisons (poison ivy or poison oak reaction), exposure to toxic chemicals, radiation damage and exposure to air borne and water borne irritant substances, which cause damaging inflammation. In addition, it is applicable to inflammatory, allergic and T cell-mediated disorders of internal organs, such as kidney, liver, heart, etc.

Dwg.0/1

L21 ANSWER 27 OF 44 WPIDS (C) 2002 THOMSON DERWENT
AN 2001-244560 [25] WPIDS
DNC C2001-073385
TI Composition comprising interleukin-12 p40 and IL-B30 polypeptide or its segment, useful for ameliorating **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor.
DC B04 D16
IN DE WAAL MALEFYT, R; KASTELEIN, R A; LIRA, S A; NARULA, S K; OPPMANN, B; RENNICK, D M; WIEKOWSKI, M T
PA (SCHE) SCHERING CORP
CYC 92
PI WO 2001018051 A2 20010315 (200125)* EN 69p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CZ DE DK DM DZ EE ES FI GB GD GE HR HU ID IL IN IS JP KG KR KZ LC LK LR LT LU LV MA MD MG MK MN MX MZ NO NZ PL PT RO RU SE SG SI SK SL TJ TM TR TT TZ UA UZ VN YU ZA
AU 2000073608 A 20010410 (200137)
EP 1210434 A2 20020605 (200238) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
ADT WO 2001018051 A2 WO 2000-US24686 20000908; AU 2000073608 A AU 2000-73608 20000908; EP 1210434 A2 EP 2000-961688 20000908, WO 2000-US24686 20000908

FDT AU 2000073608 A Based on WO 200118051; EP 1210434 A2 Based on WO 200118051
PRAI US 1999-164616P 19991110; US 1999-393090 19990909
AB WO 200118051 A UPAB: 20010508

NOVELTY - A composition (I) comprising a substantially pure polypeptide comprising a number of distinct segments of at least 7 contiguous amino acids from interleukin (**IL**)-**12** p40 and/or IL-B30, and a substantially pure polypeptide comprising a segment of at least 11 contiguous amino acids from **IL-12** p40 and/or IL-B30.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated or recombinant nucleic acid (II) encoding (I);

(2) a cell (III) comprising (II);

(3) a nucleic acid (IV) which hybridizes under wash conditions of 30 minutes at 50 deg. C and less than 1M salt to the natural mature coding portion of primate **IL-12** p40 and IL-B30;

(4) an **antagonist** (V) of **IL-12** p40/IL-B30 combined with a tumor necrosis factor-alpha (TNF alpha) **antagonist**, an **IL-12 antagonist**, IL-10, or steroids;

(5) a binding compound (VI) comprising an antigen binding site from an **antibody**, which specifically binds to (I) and comprising a substantially pure polypeptide comprising **IL-12** p40 and IL-B30 polypeptide, or a polypeptide comprising **IL-12** p40 fused to IL-B30, but not to either **IL-12** p40 or IL-B30 polypeptide;

(6) a kit (VII) comprising:

(a) (I), and a compartment comprising the polypeptide, or instructions for use or disposal of reagents in the kit;

(b) (II), and a compartment comprising (II), a compartment further comprising a primate **IL-12** p40 or IL-B30, or instructions for use or disposal of reagents in the kit or (VI); and

(c) a compartment comprising (VI), or instructions for use or disposal of reagents in the kit;

(7) producing (M1) an antigen:**antibody** complex, involves contacting, under appropriate conditions, a primate **IL-12** p40/IL-B30 composition with (VI), allowing the complex to form;

(8) a composition (VIII) comprising (VI) which is sterile, or (VI) and a carrier such as an aqueous compound, including water, saline, and/or buffer;

(9) increasing (M2) the secretion of a primate IL-B30 involves expressing the polypeptide with **IL-12** p40 or increasing the secretion of a primate **IL-12** p40 involves expressing the **IL-12** p40 with IL-B30; and

(10) screening (M3) for a receptor which binds (I) involves contacting the complex to a cell expressing the receptor under conditions allowing the complex to bind to the receptor, forming a detectable interaction.

ACTIVITY - Antirheumatic; antiarthritic; osteopathic; antiarthritic; neuroprotective; antiarteriosclerotic; cerebroprotective; vasotropic; cytostatic; antitumor; immunosuppressive.

MECHANISM OF ACTION - Modulator of physiology or development of cell in host; inducer of memory T-cell proliferation (claimed); modulator of trafficking or activation of leukocyte.

No supporting data is given.

USE - (I) is useful for modulating physiology or development of a cell or tissue in a host organism by contacting the cell with (I) or (V), resulting in an increased or decreased production of Interferon-gamma (IFN gamma), an enhanced Th1 response such as anti-tumor effect, adjuvant effect, anti-viral effect or antagonized allergic effect, and amelioration of an autoimmune condition or a chronic inflammatory condition. The contacting is in combination with IL-18, **IL-12**, radiation therapy or chemotherapy, an immune adjuvant or an anti-viral

therapeutic. The **antagonist** is an **antibody** against **IL-12** receptor subunit beta 1. The **antagonist** or agonist of mammalian IL-B30 protein is useful for modulating the inflammatory response in an animal, by contacting cells in the animal with the agonist or **antagonist**, where the animal exhibits signs or symptoms of an acute phase inflammatory response in skin, lung, gastrointestinal, or liver tissue. The modulation is accelerating maturation of neutrophils into platelets and has an effect on immunoglobin A and G (IgA and IgG) . The **antagonist** is an **antibody** which binds to the mammalian IL-B30 or blocks signaling mediated by mammalian IL-B30. The **antagonist** or agonist is administered in combination with an anti-inflammatory cytokine agonist or **antagonist**, an analgesic, an anti-inflammatory agent, or a steriod. IL-B30 or its agonist is useful inducing the proliferation of memory T-cells (all claimed).

Agonist or **antagonist** of IL-B30 protein is useful for modulating the trafficking or activation of a leukocyte in an animal experiencing science or symptoms of autoimmunity, an inflammatory condition, tissue specific autoimmunity, degenerative autoimmunity, **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis, delayed hypersensitivities, skin grafting, a transplant, spinal injury, stroke, neurodegeneration, an infectious disease, ischemia, cancer, tumors, multiple myeloma, Castleman's disease, postmenopausal osteoporosis or IL-6-associated diseases.

IL-12 p40/IL-B30 is useful as an immunogen for the production a antisera or **antibodies** specific for binding. (I) is useful for in vitro assays, scientific research, and the synthesis or manufacture of nucleic acids or **antibodies**. (II) is useful in forensic science.

Dwg.0/0

L21 ANSWER 28 OF 44 WPIDS (C) 2002 THOMSON DERWENT
AN 2001-515399 [57] WPIDS
DNC C2001-154207
TI New substituted glutarimide derivatives are **IL-12 antagonists**, are useful as immunomodulators and for the treatment of angiopathy, hematological or oncological disorders.
DC B03
IN BUSCHMANN, H; FROSCH, S; GERMANN, T; WADE, E; ZIMMER, O
PA (CHEF) GRUENENTHAL GMBH
CYC 94
PI DE 10002509 A1 20010726 (200157)* 8p
WO 2001053261 A1 20010726 (200157) DE
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001025140 A 20010731 (200171)
ADT DE 10002509 A1 DE 2000-10002509 20000121; WO 2001053261 A1 WO 2001-EP155 20010109; AU 2001025140 A AU 2001-25140 20010109
FDT AU 2001025140 A Based on WO 200153261
PRAI DE 2000-10002509 20000121
AB DE 10002509 A UPAB: 20011005
NOVELTY - Substituted glutarimide derivatives (I) are new.

DETAILED DESCRIPTION - Substituted glutarimide derivatives of formula (I) are new.

R1, R2 = COOR5, COR5 or CONR6R7, H, Cl, F, OH, NO2, NH2, 1-6C alkyl, 1-6C alkoxy, CHF2, CH2F, CF3 or an optionally substituted condensed benzol ring;

R5 = 1-6C alkyl or 3-7C cycloalkyl;

R6, R7 = 1-6C alkyl, or R6+R7 together form pyrrolidine, piperidine, hexamethyleneimine, morpholine, thiomorpholine, piperazine or N-methylpiperazine;
R3 = H, OH or CH2NR6R7;
R4 = H, 1-3C alkyl, F, CF3 or CHF2;
X = (CH2)n-NR8, (CH2)n-S, (CH2)q, C equivalent to C-(CH2)m or CH=CH-(CH2)m;
R8 = H, 1-6C alkyl, benzyl or phenylethyl (optionally substituted by Cl, F, CHF2, CH2F, CF3, OH, NO2, NH2, 1-6C alkyl or 1-6C alkoxy);
n = 1-3;
q = 2-4; and
m = 1 or 2.
Any available heteroatoms (on X) are bound to the glutarimide moiety and any methylene groups may be substituted by 1-3C alkyl, 1-3C alkoxy, F, Cl, CF3 or OH. Provided that (a) R1 and R2 are not both H and (b) R1 and R2 cannot be OCH3 when X = (CH2)n-S.
ACTIVITY - Immunomodulator; cytostatic; immunosuppressive; antiinflammatory; dermatological; antipsoriatic; antiasthmatic; antiulcer; hepatotropic; nephrotropic; antiallergic; antirheumatic; antiarthritic; neuroprotective; antidiabetic; antibacterial; antiarteriosclerotic; ophthalmological.
No biological data given.
MECHANISM OF ACTION - Interleukin-**antagonist**-12; HLA-**antagonist**.
USE - The compounds are useful as **IL-12** inhibitors with activity as immunomodulators and for the treatment of angiopathy, and hematological and oncological disorders (claimed). Non-claimed uses include the treatment of autoimmune disease, atopic dermatitis, psoriasis, eczema, bronchitis, pneumonia, bronchial asthma, ARDS, sarcoidosis, silicosis, fibrosis, gastroduodenal ulcers, Crohn's disease, ulcerative colitis, hepatitis, pancreatitis, appendicitis, peritonitis, nephritis, aphthosis, conjunctivitis, keratitis, uveitis, rhinitis, **rheumatoid arthritis**, HLA-B27 associated diseases, multiple sclerosis, juvenile onset diabetes, lupus erythematosus, sepsis, bacterial meningitis, cachexia, transplant rejection, graft-versus-host reactions, atherosclerosis, macular degeneration and diabetic retinopathy.
ADVANTAGE - Improved activity and stability against hydrolysis.
Dwg.0/0

L21 ANSWER 29 OF 44 BIOTECHDS COPYRIGHT 2002 THOMSON DERWENT AND ISI
AN 2001-08257 BIOTECHDS
TI Composition containing interleukin-12 p40 and IL-B30 protein or its segment, useful for ameliorating **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor;
vector-mediated gene transfer and expression in host cell, **antibody** and **antagonist**
AU Oppmann B; De Waal Malefyt R; Rennick D M; Kastelein R A; Wiekowski M T; Lira S A; Narula S K
PA Schering-USA
LO Kenilworth, NJ, USA.
PI WO 2001018051 15 Mar 2001
AI WO 2000-US24686 8 Sep 2000
PRAI US 1999-164616 10 Nov 1999; US 1999-393090 9 Sep 1999
DT Patent
LA English
OS WPI: 2001-244560 [25]
AB A composition containing a substantially pure protein containing a number of distinct segments of at least 7 contiguous amino acids from interleukin (**IL**)-**12** p40 and/or IL-B30, and a substantially pure protein containing a segment of at least 11 contiguous

amino acids from **IL-12** p40 and/or IL-B30, is new.
Also claimed are: a recombinant nucleic acid encoding the protein; a cell containing the nucleic acid; a nucleic acid which hybridizes under wash conditions of 30 min at 50 deg and less than 1M salt to the natural mature coding portion of primate **IL-12** p40 and IL-B30; an **antagonist** of **IL-12** p40/IL-B30 combined with a tumor necrosis factor-alpha (TNF-alpha) **antagonist**, an **IL-12 antagonist**, IL-10 or steroids; a binding compound containing an antigen binding site from an **antibody** which specifically binds to the protein; a kit containing the composition, polynucleotide and a binding compound; producing an antigen:**antibody** complex; a composition containing a binding compound; increasing the secretion of a primate IL-B30; and screening for a receptor which binds the composition. The composition is useful for modulating physiology or development of a cell or tissue0. (69pp)

L21 ANSWER 30 OF 44 USPATFULL
AN 2001:221075 USPATFULL
TI Retinoid antagonists and use thereof
IN Bollag, Werner, Basel, Switzerland
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of
Mohr, Peter, Basel, Switzerland
Panina-Bordignon, Paola, Milan, Italy
Rosenberger, Michael, Caldwell, NJ, United States
Sinigaglia, Francesco, Milan, Italy
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6326397 B1 20011204
AI US 1999-307009 19990507 (9)
RLI Continuation-in-part of Ser. No. US 1998-189189, filed on 10 Nov 1998
DT Utility
FS GRANTED
EXNAM Primary Examiner: Killos, Paul J.
LREP Johnston, George W., Parise, John P.
CLMN Number of Claims: 16
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 1573
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel retinoid antagonists of the formula I ##STR1##

wherein the dotted bond can be either hydrogenated or form a double bond; and, when the dotted bond forms a double bond, R.sup.1 is lower alkyl and R.sup.2 is hydrogen; and, when the dotted bond is hydrogenated, R.sup.1 and R.sup.2 taken together are methylene to form a cis-substituted cyclopropyl ring; R.sup.3 is hydroxy or lower alkoxy; R.sup.4 is alkyl or alkoxy; and R.sup.5 and R.sup.6 are, independently, a C.sub.4-12 alkyl or a 5-12 cycloalkyl substituent containing from 1-3 rings which are either unsubstituted or substituted with from 1-3 lower alkyl groups, with the carbon atom of R.sup.5 and R.sup.6 being linked to the remainder of the molecule to form a quaternary carbon atom pharmaceutically acceptable salts of carbocylic acids of the formula I; as well as method for the treatment of osteoporosis and preneoplastic and neoplastic diseases, and a method for reducing or abolishing adverse events in subjects receiving retinoid agonist treatment by administering a retinoid **antagonist**.

L21 ANSWER 31 OF 44 USPATFULL
AN 2001:63494 USPATFULL
TI **Antibodies** against human **IL-12**
IN Gately, Maurice Kent, Parsippany, NJ, United States

Presky, David Howard, Glen Ridge, NJ, United States
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6225117 B1 20010501
AI US 1999-232522 19990119 (9)
PRAI US 1998-72333P 19980123 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: DiBrino, Marianne
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1122
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel p75 heterodimer specific anti-human **IL-12 antibodies** that are characterized by a higher potency and greater efficacy in neutralizing human **IL-12** bioactivity than known heterodimer specific **IL-12** monoclonal **antibodies**. The heterodimer specific **antibodies** recognize one or more **epitopes** of the human **IL-12** p75 heterodimer, but do not bind to the p40 subunit alone. The heterodimer specific **IL-12 antibodies** neutralize rhesus monkey **IL-12** bioactivity with a potency similar to their potency for neutralizing human **IL-12** bioactivity making them useful **IL-12 antagonists** for in vivo studies in the rhesus monkey.

L21 ANSWER 32 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3
AN 2001:290669 BIOSIS
DN PREV200100290669
TI Antigen-specific T cell-mediated gene therapy in collagen-induced arthritis.
AU Nakajima, Atsuo; Seroogy, Christine M.; Sandora, Matthew R.; Tarner, Ingo H.; Costa, Gina L.; Taylor-Edwards, Cariel; Bachmann, Michael H.; Contag, Christopher H.; Fathman, C. Garrison (1)
CS (1) Department of Medicine, Division of Immunology and Rheumatology, School of Medicine, Stanford University, CCSR Building, Room 2225, Stanford, CA, 94305-5111: cfathman@leland.stanford.edu USA
SO Journal of Clinical Investigation, (May, 2001) Vol. 107, No. 10, pp. 1293-1301. print.
ISSN: 0021-9738.
DT Article
LA English
SL English
AB Autoantigen-specific T cells have tissue-specific homing properties, suggesting that these cells may be ideal vehicles for the local delivery of immunoregulatory molecules. We tested this hypothesis by using type II collagen-specific (CII-specific) CD4+ T hybridomas or primary CD4+ T cells after gene transfer, as vehicles to deliver an immunoregulatory protein for the treatment of collagen-induced arthritis (CIA), a mouse model of **rheumatoid arthritis** (**RA**). CII-specific T cells or hybridomas were transduced using retroviral vectors to constitutively express the **IL-12 antagonist**, **IL-12** p40. Transfer of engineered CD4+ T cells after immunization significantly inhibited the development of CIA, while cells transduced with vector control had no effect. The beneficial effect on CIA of **IL-12** p40-transduced T cells required TCR specificity against CII, since transfer of T cells specific for another antigen producing equivalent amounts of **IL-12** p40 had

no effect. In vivo cell detection using bioluminescent labels and RT-PCR showed that transferred CII-reactive T-cell hybridomas accumulated in inflamed joints in mice with CIA. These results indicate that the local delivery of **IL-12** p40 by T cells inhibited CIA by suppressing autoimmune responses at the site of inflammation. Modifying antigen-specific T cells by retroviral transduction for local expression of immunoregulatory proteins thus offers a promising strategy for treating **RA**.

L21 ANSWER 33 OF 44 USPATFULL
AN 2000:138395 USPATFULL
TI Treatment of T-helper cell type 2-mediated immune disease by retinoid antagonists
IN Bollag, Werner, Basel, Switzerland
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of
Panina-Bordignon, Paola, Milan, Italy
Sinigaglia, Francesco, Milan, Italy
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6133309 20001017
AI US 1998-189189 19981110 (9)
PRAI EP 1997-119776 19971112
DT Utility
FS Granted
EXNAM Primary Examiner: Travers, Russell
LREP Johnston, George W., Epstein, William H., Parise, John P.
CLMN Number of Claims: 37
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 780
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Retinoids with retinoid receptor antagonistic activity, pharmaceutically acceptable salts and pharmaceutically acceptable hydrolyzable esters thereof, have been found efficacious in treating T-helper cell type 2 (Th2)-mediated immune diseases, such as immunoglobulin E (IgE)-mediated allergic diseases.

L21 ANSWER 34 OF 44 USPATFULL
AN 2000:50737 USPATFULL
TI Methods and compositions for modulating responsiveness to corticosteroids
IN Sekut, Les, Westborough, MA, United States
Carter, Adam, Newburyport, MA, United States
Ghayur, Tariq, Grafton, MA, United States
Banerjee, Subhashis, Shrewsbury, MA, United States
Tracey, Daniel E., Harvard, MA, United States
PA BASF Aktiengesellschaft, Rheinland Pfalz, Germany, Federal Republic of (non-U.S. corporation)
PI US 6054487 20000425
AI US 1997-820692 19970318 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Jarvis, William R. A.
LREP Lahive & Cockfield, LLP
CLMN Number of Claims: 46
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 2404
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Method for modulating responsiveness to corticosteroids in a subject are provided. In the method of the invention, an agent which antagonizes a factor that regulates production of IFN-.gamma. in the subject is administered to the subject in combination with a corticosteroid such

that responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject. In one embodiment, the agent is an interferon-.gamma. inducing factor (IGIF) **antagonist**. In another embodiment, the agent is an interleukin-12 (**IL-12**) **antagonist**. In a preferred embodiment, the agent is an inhibitor of a caspase family protease, preferably an ICE inhibitor. In another preferred embodiment, the agent is an anti-**IL-12** monoclonal **antibody**. Other preferred agents include phosphodiesterase IV inhibitors and beta-2 agonists. The methods of the invention can be used in the treatment of a variety of inflammatory and immunological diseases and disorders. Pharmaceutical compositions comprising an agent which antagonizes a factor that regulates production of IFN-.gamma. in a subject, a corticosteroid and a pharmaceutically acceptable carrier are also provided. A preferred composition comprises an ICE inhibitor, a corticosteroid and a pharmaceutically acceptable carrier.

L21 ANSWER 35 OF 44 CAPLUS COPYRIGHT 2002 ACS
AN 1999:487326 CAPLUS
DN 131:129052
TI **Antibodies** against human **IL-12**
IN Gately, Maurcie Kent; Presky, David Howard
PA F.Hoffmann-La Roche A.-G., Switz.
SO PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9937682	A2	19990729	WO 1999-EP202	19990115
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AU 9925177	A1	19990115	AU 1999-25177	19990115
	CA 2318052	AA	19990729	CA 1999-2318052	19990115
	BR 9907743	A	20001017	BR 1999-7743	19990115
	EP 1049717	A2	20001108	EP 1999-904780	19990115
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI				
	JP 2002501085	T2	20020115	JP 2000-528602	19990115
	US 6225117	B1	20010501	US 1999-232522	19990119
	ZA 9900452	A	19990723	ZA 1999-452	19990121
PRAI	US 1998-72333P	P	19980123		
	WO 1999-EP202	W	19990115		

AB The present invention relates to p75 heterodimer specific anti-human **IL-12 antibodies** that are characterized by a higher potency and greater efficacy in neutralizing human **IL-12** bioactivity than known heterodimer specific **IL-12** monoclonal **antibodies**. The heterodimer specific **antibodies** recognize one or more **epitopes** of the human **IL-12** p75 heterodimer, but do not bind to the p40 subunit alone. The heterodimer specific **IL-12 antibodies** neutralize rhesus monkey **IL-12** bioactivity with a potency similar to their potency for neutralizing human **IL-12** bioactivity making them useful **IL-12 antagonists**. The monoclonal **antibodies** are therefore useful for diseases assocd. with aberrant Th1-type helper cell

activity, e.g. multiple sclerosis, **rheumatoid arthritis**, autoimmune diabetes mellitus, Crohn's disease and ulcerative colitis.

L21 ANSWER 36 OF 44 USPATFULL
AN 1999:43184 USPATFULL
TI Membrane-bound cytokine compositions comprising GM=CSF and methods of modulating an immune response using same
IN Hoo, William Soo, Carlsbad, CA, United States
PA The Immune Response Corporation, Carlsbad, CA, United States (U.S. corporation)
PI US 5891432 19990406
AI US 1997-902516 19970729 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Spector, Lorraine
LREP Campbell & Flores LLP
CLMN Number of Claims: 24
ECL Exemplary Claim: 1,13
DRWN 9 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1917
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule such as GM-CSF operatively fused to a heterologous membrane attachment domain. Non-**antibody** immunomodulatory molecules useful in the invention include immunostimulatory and immunosuppressive molecules such as cytokines. In one embodiment, the invention provides a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule operatively fused to a heterologous membrane attachment domain and, additionally, a disease-associated antigen or immunogenic **epitope** thereof. Further provided by the invention are methods of modulating an immune response against a disease-associated antigen by administering to an individual a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule operatively fused to a heterologous membrane attachment domain.

L21 ANSWER 37 OF 44 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 4
AN 1998-520957 [44] WPIDS
DNC C1998-156445
TI Modulating responsiveness to corticosteroid e.g. in treating auto-immune diseases - by administering agent antagonising target that regulates production of interferon gamma.
DC B01 B04 B05
IN BANERJEE, S; CARTER, A; GHAYUR, T; SEKUT, L; TRACEY, D E
PA (BADI) BASF AG
CYC 81
PI WO 9841232 A2 19980924 (199844)* EN 112p
RW: AT BE CH DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE GH GM GW HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW
AU 9867604 A 19981012 (199907)
NO 9904506 A 19991117 (200005)
CZ 9903127 A3 20000315 (200021)
EP 998300 A1 20000510 (200027) EN
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE
US 6054487 A 20000425 (200027)
ES 2146192 T1 20000801 (200040)

BR 9810409 A 20000822 (200050)
CN 1269722 A 20001011 (200103)
SK 9901221 A3 20001211 (200103)
MX 9908433 A1 19991201 (200110)
KR 2000076420 A 20001226 (200134)
AU 734756 B 20010621 (200141)
JP 2002504091 W 20020205 (200212) 154p
HU 2001004439 A2 20020429 (200238)

ADT WO 9841232 A2 WO 1998-US4916 19980312; AU 9867604 A AU 1998-67604 19980312; NO 9904506 A WO 1998-US4916 19980312, NO 1999-4506 19990917; CZ 9903127 A3 WO 1998-US4916 19980312, CZ 1999-3127 19980312; EP 998300 A1 EP 1998-912929 19980312, WO 1998-US4916 19980312; US 6054487 A US 1997-820692 19970318; ES 2146192 T1 EP 1998-912929 19980312; BR 9810409 A BR 1998-10409 19980312, WO 1998-US4916 19980312; CN 1269722 A CN 1998-805124 19980312; SK 9901221 A3 WO 1998-US4916 19980312, SK 1999-1221 19980312; MX 9908433 A1 MX 1999-8433 19990914; KR 2000076420 A WO 1998-US4916 19980312, KR 1999-708524 19990918; AU 734756 B AU 1998-67604 19980312; JP 2002504091 W JP 1998-540633 19980312, WO 1998-US4916 19980312; HU 2001004439 A2 WO 1998-US4916 19980312, HU 2001-4439 19980312

FDT AU 9867604 A Based on WO 9841232; CZ 9903127 A3 Based on WO 9841232; EP 998300 A1 Based on WO 9841232; ES 2146192 T1 Based on EP 998300; BR 9810409 A Based on WO 9841232; KR 2000076420 A Based on WO 9841232; AU 734756 B Previous Publ. AU 9867604, Based on WO 9841232; JP 2002504091 W Based on WO 9841232; HU 2001004439 A2 Based on WO 9841232

PRAI US 1998-16346 19980130; US 1997-820692 19970318

AB WO 9841232 A UPAB: 19981104

Modulating responsiveness to corticosteroids comprises administering: (a) an agent which antagonises a target that regulates production of interferon- gamma (IFN- gamma), to inhibit production of IFN- gamma and (b) a corticosteroid.

Preferably, the agent which antagonises a target that regulates production of IFN- gamma is an IL-18 **antagonist** e.g. an inhibitor of a caspase family protease (especially an ICE inhibitor) or an **antibody** (**fragment**) or engineered binding protein that binds IL-18 or an IL-18 receptor. The agent may also be an **IL-12 antagonist** e.g. an agent that stimulates cyclic AMP production in cells that produce **IL-12**, especially a phosphodiesterase IV inhibitor such as a 4-arylpyrrolidinone, rolipram, denbufylline, tibenelast, nitraquazone, CP-80633, CP-77059 or a quinazolinedione or a beta -2 agonist such as salmeterol, fenoterol or isoproterenol.

USE- The process is used for treating septic shock, Crohn's disease, asthma, graft versus host disease or transplant rejection autoimmune disease or disorder and immunoinflammatory diseases or disorders comprising adult respiratory distress syndrome, systemic lupus erythematosus, inflammatory bowel disease, ulcerative colitis, multiple sclerosis, insulin dependent diabetes mellitus, **rheumatoid arthritis**, juvenile **rheumatoid arthritis**, psoriatic arthritis, inflammatory pulmonary syndrome, pemphigus vulgaris, idiopathic thrombocytopenic purpura, autoimmune meningitis, myasthenia gravis, autoimmune thyroiditis, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, Sjogren's syndrome, keratoconjunctivitis, cutaneous lupus erythematosus, scleroderma, vaginitis, proctitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, erythema nodosum leprosum, autoimmune uveitis, allergic encephalomyelitis, aplastic anaemia, pure red cell anaemia, idiopathic thrombocytopenia, polychondritis, Wegener's granulomatosis, chronic active hepatitis, Graves ophthalmopathy,primary biliary cirrhosis, uveitis posterior and interstitial lung fibrosis. Administration is oral, intravenous or ophthalmic.

ADVANTAGE - The process reverses steroid resistance and increases steroid sensitivity.

AB The present invention relates to a novel **antibody** against the **IL-12** receptor and a novel combination of anibodies anainst the **IL-12** receptor. The novel anti-**IL-12** receptor anbody, designated as 2B10, provided in accordance with the present invention binds to the human **IL-12** receptor but which is not capable of inhibiting the binding of human **IL-12** to the high affinity human **IL-12** receptor and is not capable of neutralizing human **IL-12** bioactivity by binding to human **IL-12** receptor.

L21 ANSWER 40 OF 44 USPATFULL
AN 1998:135151 USPATFULL
TI Human receptor for interleukin-12
IN Chua, Anne On, Wayne, NJ, United States
Gubler, Ulrich Andreas, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5831007 19981103
AI US 1995-419652 19950411 (8)
RLI Division of Ser. No. US 1994-248532, filed on 31 May 1994, now patented, Pat. No. US 5536657 which is a continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John
LREP Johnston, George W., Epstein, William H., Bucholz, Briana C.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 35 Drawing Figure(s); 26 Drawing Page(s)
LN.CNT 1937
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to substantially pure Interleukin-12 receptor cDNAs and protein and uses therefore. The Interleukin-12 receptor is shown to be a member of the cytokine receptor superfamily and has a high homology to human gp130.

L21 ANSWER 41 OF 44 WPIDS (C) 2002 THOMSON DERWENT
AN 1997-147515 [14] WPIDS
DNN N1997-122015 DNC C1997-047130
TI New interleukin-12 beta-2 receptor and high binding affinity complexes - have a high affinity for interleukin-12, and are used to treat auto immune diseases.
DC B04 D16 S03
IN GUBLER, U A; PRESKY, D H
PA (HOFF) HOFFMANN LA ROCHE & CO AG F; (HOFF) HOFFMANN LA ROCHE INC
CYC 20
PI EP 759466 A2 19970226 (199714)* EN 53p
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
JP 09132598 A 19970520 (199730) 43p
EP 759466 A3 19970528 (199732)
US 5840530 A 19981124 (199903)
US 5852176 A 19981222 (199907)#
US 5919903 A 19990706 (199933)
JP 2948150 B2 19990913 (199943) 43p
ADT EP 759466 A2 EP 1996-111807 19960723; JP 09132598 A JP 1996-196385 19960725; EP 759466 A3 EP 1996-111807 19960723; US 5840530 A Provisional US 1995-1701P 19950801, Provisional US 1996-18674P 19960530, US 1996-685118 19960723; US 5852176 A Div ex US 1996-685118 19960723, US 1997-915495 19970820; US 5919903 A Provisional US 1995-1701P 19950801, Provisional US 1996-18674P 19960530, Div ex US 1996-685118 19960723, US 1997-914520 19970819; JP 2948150 B2 JP 1996-196385 19960725
FDT JP 2948150 B2 Previous Publ. JP 09132598

PRAI US 1996-18674P 19960530; US 1995-1701P 19950801; US 1996-685118 19960723; US 1997-915495 19970820; US 1997-914520 19970819

AB EP 759466 A UPAB: 19970407
A novel low binding affinity (BA) interleukin-12 (**IL-12**) beta 2 receptor protein (A), or a **fragment**, has a low BA for **IL-12**, and when complexed with an **IL-12** beta 1 receptor protein (B), forms a complex having a high BA for **IL-12**. Also new are: (1) a complex with a high BA for **IL-12**, comprising (A) or a **fragment**, complexed with **IL-12** beta 1 receptor protein, or a **fragment**, which has a low BA for **IL-12**, and, when complexed with (A), has a high BA for **IL-12**; (2) a protein encoded by first and second nucleic acids, the first comprising two subsequences (SS), where one SS encodes a soluble **fragment** of (A), and the other SS (SS2) encodes all the domains of the constant region of the heavy chain of human Ig, except the first domain of the constant region, and the second nucleic acid has two SS, where one SS encodes a soluble **fragment** of (B) and the other SS is as for SS2; (3) nucleic acids encoding the proteins or complexes; (4) vectors contg. the nucleic acid of (3); (5) host cells transformed with the nucleic acid of (3); and (6) **antibodies** against (A) or (B).
USE - The proteins, complexes or **antibodies** may be used in therapeutic compsns., pref. with at least 1 cytokine antagonists (claimed). The compsns. are used to treat autoimmune dysfunctions (claimed), such as **rheumatoid arthritis**, inflammatory bowel disease and multiple sclerosis. The proteins or complexes can also be used to detect **antagonists** and agonists of **IL-12** activity (claimed).
Dwg.0/6

L21 ANSWER 42 OF 44 USPATFULL
AN 96:63048 USPATFULL
TI Recombinant DNA encoding human receptor for interleukin-12
IN Chua, Anne O., Wayne, NJ, United States
Gubler, Ulrich A., Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5536657 19960716
AI US 1994-248532 19940531 (8)
RLI Continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John
LREP Gould, George M., Johnston, George W., Kass, Alan P.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 34 Drawing Figure(s); 25 Drawing Page(s)
LN.CNT 1755
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to substantially pure Interleukin-12 receptor cDNAs and protein and uses therefore. The Interleukin-12 receptor is shown to be a member of the cytokine receptor superfamily and has a high homology to human gp130.

L21 ANSWER 43 OF 44 CAPLUS COPYRIGHT 2002 ACS
AN 1995:934127 CAPLUS
DN 123:337469
TI Use of **IL-12** and **IL-12** **antagonists** in treatment of autoimmune diseases
IN Leonard, John P.; Goldman, Samuel; O'Hara, Richard, Jr.
PA Genetics Institute, Inc., USA
SO PCT Int. Appl., 37 pp.

CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9524918	A1	19950921	WO 1995-US2550	19950307
	W: AU, CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	ZA 9500960	A	19951010	ZA 1995-960	19950207
	TW 400233	B	20000801	TW 1995-84101380	19950214
	IL 112677	A1	20000131	IL 1995-112677	19950216
	CA 2185565	AA	19950921	CA 1995-2185565	19950307
	AU 9519749	A1	19951003	AU 1995-19749	19950307
	AU 689236	B2	19980326		
	EP 750509	A1	19970102	EP 1995-912666	19950307
	EP 750509	B1	20020515		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
	JP 09510444	T2	19971021	JP 1995-524044	19950307
	EP 1179348	A2	20020213	EP 2001-117762	19950307
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE				
	US 6338848	B1	20020115	US 2000-513380	20000225
PRAI	US 1994-212629	A	19940314		
	EP 1995-912666	A3	19950307		
	WO 1995-US2550	W	19950307		
	US 1995-560943	B1	19951120		

AB Autoimmune conditions such as multiple sclerosis, systemic lupus erythematosus, **rheumatoid arthritis**, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin-dependent diabetes mellitus, and autoimmune inflammatory eye disease, esp. conditions which are promoted by an increase in levels of IFN-.gamma. or TNF-.alpha., are treated in mammals by administering **IL-12** or an **IL-12 antagonist** . Thus, lymphocytes from mice immunized with myelin proteolipid protein, and restimulated with a synthetic peptide from this protein, were injected into naive mice. The injected mice developed exptl. allergic encephalomyelitis which was exacerbated by incubation of these lymphocytes with **IL-12** during restimulation, and alleviated by injection of a polyclonal **antibody** to **IL-12**.

L21 ANSWER 44 OF 44 MEDLINE
AN 94032770 MEDLINE
DN 94032770 PubMed ID: 8218599
TI Clinical and preclinical studies presented at the Keystone Symposium on Arthritis, Related Diseases, and Cytokines.
AU Ralph P
CS Department of Immunology, Genentech, Inc., South San Francisco, CA 94080.
SO LYMPHOKINE AND CYTOKINE RESEARCH, (1993 Aug) 12 (4) 261-3.
Journal code: 9107882. ISSN: 1056-5477.
CY United States
DT Conference; Conference Article; (CONGRESSES)
LA English
FS Priority Journals
EM 199312
ED Entered STN: 19940117
Last Updated on STN: 19990129
Entered Medline: 19931203
AB Topics include treatment of multiple sclerosis (MS) with T cell receptor (TCR) peptides, **rheumatoid arthritis** (**RA**) with IL-1ra, IL-2 toxin conjugate, or **antibodies** to TNF, to CD4, or to ICAM-1, sepsis and five other diseases with IL-1ra, and treatment of experimental animal diseases with soluble receptors, **IL-**

12, TGF-beta2, or small molecule **antagonists** of cytokines.

=> d clm 1 14
NO VALID FORMATS ENTERED FOR FILE 'BIOSIS'
In a multifile environment, each file must have at least one valid format requested. Refer to file specific help messages or the STNGUIDE file for information on formats available in individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):.

L21 ANSWER 1 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
AN 2002:166945 BIOSIS
DN PREV200200166945
TI Use of **IL-12** and **IL-12** **antagonists** in the treatment of autoimmune diseases.
AU Leonard, John (1); Goldman, Samuel; O'Hara, Richard, Jr.
CS (1) Auburn, NH USA
ASSIGNEE: Genetics Institute, Inc.
PI US 6338848 January 15, 2002
SO Official Gazette of the United States Patent and Trademark Office Patents, (Jan. 15, 2002) Vol. 1254, No. 3, pp. No Pagination.
http://www.uspto.gov/web/menu/patdata.html. e-file.
ISSN: 0098-1133.
DT Patent
LA English

L21 ANSWER 14 OF 44 USPATFULL
AN 2002:106416 USPATFULL
TI Nucleic acids, proteins and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PI US 2002055627 A1 20020509
AI US 2001-925299 A1 20010810 (9)
RLI Continuation of Ser. No. WO 2000-US5883, filed on 8 Mar 2000, UNKNOWN
PRAI US 1999-124270P 19990312 (60)
DT Utility
FS APPLICATION
LN.CNT 20658
INCL INCLM: 536/023.500
INCLS: 435/320.100; 435/325.000; 530/324.000; 530/387.900; 514/002.000; 435/007.200
NCL NCLM: 536/023.500
NCLS: 435/320.100; 435/325.000; 530/324.000; 530/387.900; 514/002.000; 435/007.200
IC [7]
ICM: A01N037-18
ICS: A61K038-00; G01N033-53; G01N033-567; C07H021-04; C12N015-00; C12N015-09; C12N015-63; C12N015-70; C12N015-74; C07K005-00; C07K007-00; C07K016-00; C07K017-00; C12N005-00; C12N005-02; C12P021-08
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d clm 14

L21 ANSWER 14 OF 44 USPATFULL
CLM What is claimed is:
1. An isolated nucleic acid molecule comprising a polynucleotide having a nucleotide sequence at least 95% identical to a sequence selected from the group consisting of: (a) a polynucleotide **fragment** of SEQ

ID NO:X or a polynucleotide **fragment** of the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X; (b) a polynucleotide encoding a polypeptide **fragment** of SEQ ID NO:Y or a polypeptide **fragment** encoded by the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X; (c) a polynucleotide encoding a polypeptide **fragment** of a polypeptide encoded by SEQ ID NO:X or a polypeptide **fragment** encoded by the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X; (d) a polynucleotide encoding a polypeptide domain of SEQ ID NO:Y or a polypeptide domain encoded by the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X; (e) a polynucleotide encoding a polypeptide **epitope** of SEQ ID NO:Y or a polypeptide **epitope** encoded by the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X; (f) a polynucleotide encoding a polypeptide of SEQ ID NO:Y or the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X, having biological activity; (g) a polynucleotide which is a variant of SEQ ID NO:X; (h) a polynucleotide which is an allelic variant of SEQ ID NO:X; (i) a polynucleotide which encodes a species homologue of the SEQ ID NO:Y; (j) a polynucleotide capable of hybridizing under stringent conditions to any one of the polynucleotides specified in (a)-(i), wherein said polynucleotide does not hybridize under stringent conditions to a nucleic acid molecule having a nucleotide sequence of only A residues or of only T residues.

2. The isolated nucleic acid molecule of claim 1, wherein the polynucleotide **fragment** comprises a nucleotide sequence encoding a protein.

3. The isolated nucleic acid molecule of claim 1, wherein the polynucleotide **fragment** comprises a nucleotide sequence encoding the sequence identified as SEQ ID NO:Y or the polypeptide encoded by the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X.

4. The isolated nucleic acid molecule of claim 1, wherein the polynucleotide **fragment** comprises the entire nucleotide sequence of SEQ ID NO:X or the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X.

5. The isolated nucleic acid molecule of claim 2, wherein the nucleotide sequence comprises sequential nucleotide deletions from either the C-terminus or the N-terminus.

6. The isolated nucleic acid molecule of claim 3, wherein the nucleotide sequence comprises sequential nucleotide deletions from either the C-terminus or the N-terminus.

7. A recombinant vector comprising the isolated nucleic acid molecule of claim 1.

8. A method of making a recombinant host cell comprising the isolated nucleic acid molecule of claim 1.

9. A recombinant host cell produced by the method of claim 8.

10. The recombinant host cell of claim 9 comprising vector sequences.

11. An isolated polypeptide comprising an amino acid sequence at least 95% identical to a sequence selected from the group consisting of: (a) a polypeptide **fragment** of SEQ ID NO:Y or of the sequence

encoded by the cDNA included in the related cDNA clone; (b) a polypeptide **fragment** of SEQ ID NO:Y or of the sequence encoded by the cDNA included in the related cDNA clone, having biological activity; (c) a polypeptide domain of SEQ ID NO:Y or of the sequence encoded by the cDNA included in the related cDNA clone; (d) a polypeptide **epitope** of SEQ ID NO:Y or of the sequence encoded by the cDNA included in the related cDNA clone; (e) a full length protein of SEQ ID NO:Y or of the sequence encoded by the cDNA included in the related cDNA clone; (f) a variant of SEQ ID NO:Y; (g) an allelic variant of SEQ ID NO:Y; or (h) a species homologue of the SEQ ID NO:Y.

12. The isolated polypeptide of claim 11, wherein the full length protein comprises sequential amino acid deletions from either the C-terminus or the N-terminus.

13. An isolated **antibody** that binds specifically to the isolated polypeptide of claim 11.

14. A recombinant host cell that expresses the isolated polypeptide of claim 11.

15. A method of making an isolated polypeptide comprising: (a) culturing the recombinant host cell of claim 14 under conditions such that said polypeptide is expressed; and (b) recovering said polypeptide.

16. The polypeptide produced by claim 15.

17. A method for preventing, treating, or ameliorating a medical condition, comprising administering to a mammalian subject a therapeutically effective amount of the polypeptide of claim 11 or the polynucleotide of claim 1.

18. A method of diagnosing a pathological condition or a susceptibility to a pathological condition in a subject comprising: (a) determining the presence or absence of a mutation in the polynucleotide of claim 1; and (b) diagnosing a pathological condition or a susceptibility to a pathological condition based on the presence or absence of said mutation.

19. A method of diagnosing a pathological condition or a susceptibility to a pathological condition in a subject comprising: (a) determining the presence or amount of expression of the polypeptide of claim in a biological sample; and (b) diagnosing a pathological condition or a susceptibility to a pathological condition based on the presence or amount of expression of the polypeptide.

20. A method for identifying a binding partner to the polypeptide of claim 1 comprising: (a) contacting the polypeptide of claim 11 with a binding partner; and (b) determining whether the binding partner effects an activity of the polypeptide.

21. The gene corresponding to the cDNA sequence of SEQ ID NO:Y.

22. A method of identifying an activity in a biological assay, wherein the method comprises: (a) expressing SEQ ID NO:X in a cell; (b) isolating the supernatant; (c) detecting an activity in a biological assay; and (d) identifying the protein in the supernatant having the activity.

23. The product produced by the method of claim 20.

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=> d his

     (FILE 'HOME' ENTERED AT 11:40:44 ON 11 JUL 2002)

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     LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:42:50 ON 11 JUL 2002
                E LEONARD JOHN P/AU
L1           108 S E3-E5
                E LEONARD J P/AU
L2           359 S E3-E4
                E GOLDMAN SAMUEL/AU
L3            78 S E2-E9
                E GOLDMAN S/AU
L4          1458 S E3
                E OHARA RICHARD/AU
                E OHARA R/AU
L5            70 S E3
L6          2059 S L1-L5
L7           109 S L6 AND (IL-12 OR RA OR ARTHRITIS)
L8            43 S L7 AND (ANTIBOD? OR ANTAGONIST?)
L9            20 DUP REM L8 (23 DUPLICATES REMOVED)
L10            1 S L9 AND P40

     FILE 'STNGUIDE' ENTERED AT 11:51:09 ON 11 JUL 2002
L11            0 S L8 AND (FRAGMENT? OR EPITOP?)
L12            2 S RA OR RHEUMATOID ARTHRITIS

     FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABA, WPIDS, JAPIO, BIOTECHDS,
     LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:55:50 ON 11 JUL 2002
L13            3 S L9 AND (FRAGMENT? OR EPITOP?)
L14       656173 S RA OR RHEUMATOID ARTHRITIS
L15         1079 S L14 AND IL-12
L16          724 S L15 AND (ANTIBOD? OR EPITOP? OR FRAGMENT? OR ANTAGONIST)
L17           95 S L16 AND IL-12 (5A) ANTIBOD?
L18           82 DUP REM L17 (13 DUPLICATES REMOVED)
L19           18 S L18 AND P40
L20           52 S L16 AND IL-12 (5A) ANTAGONIST?
L21           44 DUP REM L20 (8 DUPLICATES REMOVED)

=> s l21 and p40
  11 FILES SEARCHED...
L22           11 L21 AND P40

=> d bib 1-11

L22  ANSWER 1 OF 11  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN   2001:290669  BIOSIS
DN   PREV200100290669
TI   Antigen-specific T cell-mediated gene therapy in collagen-induced
     arthritis.
AU   Nakajima, Atsuo; Seroogy, Christine M.; Sandora, Matthew R.; Tarner, Ingo
     H.; Costa, Gina L.; Taylor-Edwards, Cariel; Bachmann, Michael H.; Contag,
     Christopher H.; Fathman, C. Garrison (1)
CS   (1) Department of Medicine, Division of Immunology and Rheumatology,
     School of Medicine, Stanford University, CCSR Building, Room 2225,
     Stanford, CA, 94305-5111: cfathman@leland.stanford.edu USA
SO   Journal of Clinical Investigation, (May, 2001) Vol. 107, No. 10, pp.
     1293-1301. print.
     ISSN: 0021-9738.
```

DT Article
LA English
SL English

L22 ANSWER 2 OF 11 WPIDS (C) 2002 THOMSON DERWENT
AN 2001-244560 [25] WPIDS
DNC C2001-073385
TI Composition comprising interleukin-12 **p40** and IL-B30 polypeptide or its segment, useful for ameliorating **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor.
DC B04 D16
IN DE WAAL MALEFYT, R; KASTELEIN, R A; LIRA, S A; NARULA, S K; OPPMANN, B; RENNICK, D M; WIEKOWSKI, M T
PA (SCHE) SCHERING CORP
CYC 92
PI WO 2001018051 A2 20010315 (200125)* EN 69p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CZ DE DK DM DZ EE ES FI GB GD GE HR HU ID IL IN IS JP KG KR KZ LC LK LR LT LU LV MA MD MG MK MN MX MZ NO NZ PL PT RO RU SE SG SI SK SL TJ TM TR TT TZ UA UZ VN YU ZA
AU 2000073608 A 20010410 (200137)
EP 1210434 A2 20020605 (200238) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
ADT WO 2001018051 A2 WO 2000-US24686 20000908; AU 2000073608 A AU 2000-73608 20000908; EP 1210434 A2 EP 2000-961688 20000908, WO 2000-US24686 20000908
FDT AU 2000073608 A Based on WO 200118051; EP 1210434 A2 Based on WO 200118051
PRAI US 1999-164616P 19991110; US 1999-393090 19990909

L22 ANSWER 3 OF 11 BIOTECHDS COPYRIGHT 2002 THOMSON DERWENT AND ISI
AN 2001-08257 BIOTECHDS
TI Composition containing interleukin-12 **p40** and IL-B30 protein or its segment, useful for ameliorating **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor;
vector-mediated gene transfer and expression in host cell, **antibody** and **antagonist**
AU Oppmann B; De Waal Malefyt R; Rennick D M; Kastelein R A; Wiekowski M T; Lira S A; Narula S K
PA Schering-USA
LO Kenilworth, NJ, USA.
PI WO 2001018051 15 Mar 2001
AI WO 2000-US24686 8 Sep 2000
PRAI US 1999-164616 10 Nov 1999; US 1999-393090 9 Sep 1999
DT Patent
LA English
OS WPI: 2001-244560 [25]

L22 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2002 ACS
AN 1999:487326 CAPLUS
DN 131:129052
TI **Antibodies** against human **IL-12**
IN Gately, Maurcie Kent; Presky, David Howard
PA F.Hoffmann-La Roche A.-G., Switz.
SO PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9937682	A2	19990729	WO 1999-EP202	19990115
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AU 9925177	A1	19990115	AU 1999-25177	19990115
	CA 2318052	AA	19990729	CA 1999-2318052	19990115
	BR 9907743	A	20001017	BR 1999-7743	19990115
	EP 1049717	A2	20001108	EP 1999-904780	19990115
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI				
	JP 2002501085	T2	20020115	JP 2000-528602	19990115
	US 6225117	B1	20010501	US 1999-232522	19990119
	ZA 9900452	A	19990723	ZA 1999-452	19990121
PRAI	US 1998-72333P	P	19980123		
	WO 1999-EP202	W	19990115		

L22 ANSWER 5 OF 11 USPATFULL
AN 2002:84902 USPATFULL
TI Nucleic acids, proteins and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PI US 2002044941 A1 20020418
AI US 2001-925302 A1 20010810 (9)
RLI Continuation-in-part of Ser. No. WO 2000-US5918, filed on 8 Mar 2000, UNKNOWN
PRAI US 1999-124270P 19990312 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 21121
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L22 ANSWER 6 OF 11 USPATFULL
AN 2001:221075 USPATFULL
TI Retinoid antagonists and use thereof
IN Bollag, Werner, Basel, Switzerland
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of
Mohr, Peter, Basel, Switzerland
Panina-Bordignon, Paola, Milan, Italy
Rosenberger, Michael, Caldwell, NJ, United States
Sinigaglia, Francesco, Milan, Italy
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6326397 B1 20011204
AI US 1999-307009 19990507 (9)
RLI Continuation-in-part of Ser. No. US 1998-189189, filed on 10 Nov 1998
DT Utility
FS GRANTED
EXNAM Primary Examiner: Killos, Paul J.
LREP Johnston, George W., Parise, John P.
CLMN Number of Claims: 16
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 1573

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L22 ANSWER 7 OF 11 USPATFULL
AN 2001:63494 USPATFULL
TI **Antibodies** against human **IL-12**
IN Gately, Maurice Kent, Parsippany, NJ, United States
Presky, David Howard, Glen Ridge, NJ, United States
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6225117 B1 20010501
AI US 1999-232522 19990119 (9)
PRAI US 1998-72333P 19980123 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: DiBrino, Marianne
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1122
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L22 ANSWER 8 OF 11 USPATFULL
AN 2000:138395 USPATFULL
TI Treatment of T-helper cell type 2-mediated immune disease by retinoid antagonists
IN Bollag, Werner, Basel, Switzerland
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of
Panina-Bordignon, Paola, Milan, Italy
Sinigaglia, Francesco, Milan, Italy
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6133309 20001017
AI US 1998-189189 19981110 (9)
PRAI EP 1997-119776 19971112
DT Utility
FS Granted
EXNAM Primary Examiner: Travers, Russell
LREP Johnston, George W., Epstein, William H., Parise, John P.
CLMN Number of Claims: 37
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 780
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L22 ANSWER 9 OF 11 USPATFULL
AN 2000:50737 USPATFULL
TI Methods and compositions for modulating responsiveness to corticosteroids
IN Sekut, Les, Westborough, MA, United States
Carter, Adam, Newburyport, MA, United States
Ghayur, Tariq, Grafton, MA, United States
Banerjee, Subhashis, Shrewsbury, MA, United States
Tracey, Daniel E., Harvard, MA, United States
PA BASF Aktiengesellschaft, Rheinland Pfalz, Germany, Federal Republic of (non-U.S. corporation)
PI US 6054487 20000425
AI US 1997-820692 19970318 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Jarvis, William R. A.
LREP Lahive & Cockfield, LLP
CLMN Number of Claims: 46

ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 2404
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L22 ANSWER 10 OF 11 USPATFULL
AN 1998:135151 USPATFULL
TI Human receptor for interleukin-12
IN Chua, Anne On, Wayne, NJ, United States
Gubler, Ulrich Andreas, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5831007 19981103
AI US 1995-419652 19950411 (8)
RLI Division of Ser. No. US 1994-248532, filed on 31 May 1994, now patented, Pat. No. US 5536657 which is a continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John
LREP Johnston, George W., Epstein, William H., Bucholz, Briana C.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 35 Drawing Figure(s); 26 Drawing Page(s)
LN.CNT 1937
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L22 ANSWER 11 OF 11 USPATFULL
AN 96:63048 USPATFULL
TI Recombinant DNA encoding human receptor for interleukin-12
IN Chua, Anne O., Wayne, NJ, United States
Gubler, Ulrich A., Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5536657 19960716
AI US 1994-248532 19940531 (8)
RLI Continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John
LREP Gould, George M., Johnston, George W., Kass, Alan P.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 34 Drawing Figure(s); 25 Drawing Page(s)
LN.CNT 1755
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d clm 8

L22 ANSWER 8 OF 11 USPATFULL
CLM What is claimed is:
1. A method of treating an immunoglobulin E-mediated allergic disease selected from the group consisting of allergic rhinitis and bronchial asthma, which comprises administering to a subject in need of such treatment having said immunoglobulin E-mediated allergic disease an effective amount of a retinoid **antagonist** of the formula: ##STR7## wherein R.sup.1 is C.sub.5-10 -alkyl, or a pharmaceutically acceptable salt of such retinoid **antagonist** or a pharmaceutically acceptable hydrolyzable ester of such retinoid **antagonist** or its salt.

2. The method of claim 1, wherein the administering comprises oral

administration.

3. The method of claim 2, wherein the oral administration is at a daily dosage of from about 0.05 mg to about 20 mg of the compound per kg of body weight of the subject.

4. The method of claim 3, wherein the oral administration is at a daily dosage of from about 0.3 mg to about 1.5 mg of the compound per kg of body weight of the subject.

5. The method of claim 2, wherein the oral administration comprises administering a tablet, capsule, pill or sachet containing from about 5 mg to about 200 mg of the compound.

6. The method of claim 5, wherein the oral administration comprises administering a tablet, capsule, pill or sachet containing from about 20 mg to about 100 mg of the compound.

7. The method of claim 1, wherein the administering comprises topical administration.

8. The method of claim 7, wherein the topical administration comprises administering an ointment, cream, lotion, or spray containing from about 0.01 percent to about 5.0 percent by weight of the compound.

9. The method of claim 8, wherein the topical administration comprises administering an ointment, cream, lotion, or spray containing from about 0.1 percent to about 1.0 percent by weight of the compound.

10. The method of claim 1, wherein the administering comprises inhalation.

11. The method of claim 10, wherein the inhalation comprises administering a nasal aerosol, aerosol for inhalation, or dry powder for inhalation containing from about 0.01 percent to about 5.0 percent by weight of the compound.

12. The method of claim 11, wherein the inhalation comprises administering a nasal aerosol, aerosol for inhalations, or dry powder for inhalation containing from about 0.1 percent to about 1.0 percent by weight of the compound.

13. The method of claim 1, wherein the compound is a retinoid antagonists or a alkali metal salt, alkaline earth metal salt, benzyl ester, lower alkyl ester, or 9-fluorenylmethyl ester thereof.

14. The method of claim 13, wherein the immunoglobulin E-mediated allergic disease is allergic rhinitis.

15. The method of claim 14, wherein the administering comprises oral administration.

16. The method of claim 15, wherein the oral administration is at a daily dosage of from about 0.05 mg to about 20 mg of the compound per kg of body weight of the subject.

17. The method of claim 16, wherein the oral administration is at a daily dosage of from about 0.3 mg to about 1.5 mg of the compound per kg of body weight of the subject.

18. The method of claim 15, wherein the oral administration comprises administering a tablet, capsule, pill or sachet containing from about 5

mg to about 200 mg of the compound.

19. The method of claim 18, wherein the oral administration comprises administering a tablet, capsule, pill or sachet containing from about 20 mg to about 100 mg of the compound.

20. The method of claim 19, wherein the topical administration comprises administering an ointment, cream, lotion, or spray containing from about 0.01 percent to about 5.0 percent by weight of the compound.

21. The method of claim 20, wherein the topical administration comprises administering an ointment, cream, lotion, or spray containing from about 0.1 percent to about 1.0 percent by weight of the compound.

22. The method of claim 14, the administering comprises inhalation.

23. The method of claim 22, wherein the inhalation comprises administering a nasal aerosol, aerosol for inhalation, or dry powder for inhalation containing from about 0.01 percent to about 5.0 percent by weight of the compound.

24. The method of claim 23, wherein the inhalation comprises administering a nasal aerosol, aerosol for inhalations, or dry powder for inhalation containing from about 0.1 percent to about 1.0 percent by weight of the compound.

25. The method of claim 14, wherein the compound is a retinoid antagonists or a alkali metal salt, alkaline earth metal salt, benzyl ester, lower alkyl ester, or 9-fluorenylmethyl ester thereof.

26. The method of claim 7, wherein the immunoglobulin E-mediated allergic disease is allergic bronchial asthma.

27. The method of claim 26, wherein the administering comprises oral administration.

28. The method of claim 27, wherein the oral administration is at a daily dosage of from about 0.05 mg to about 20 mg of the compound per kg of body weight of the subject.

29. The method of claim 28, wherein the oral administration is at a daily dosage of from about 0.3 mg to about 1.5 mg of the compound per kg of body weight of the subject.

30. The method of claim 27, wherein the oral administration comprises administering a tablet, capsule, pill or sachet containing from about 5 mg to about 200 mg of the compound.

31. The method of claim 30, wherein the oral administration comprises administering a tablet, capsule, pill or sachet containing from about 20 mg to about 100 mg of the compound.

32. The method of claim 31, wherein the topical administration comprises administering an ointment, cream, lotion, or spray containing from about 0.01 percent to about 5.0 percent by weight of the compound.

33. The method of claim 32, wherein the topical administration comprises administering an ointment, cream, lotion, or spray containing from about 0.1 percent to about 1.0 percent by weight of the compound.

34. The method of claim 26, wherein the administering comprises inhalation.

35. The method of claim 34, wherein the inhalation comprises administering a nasal aerosol, aerosol for inhalation, or dry powder for inhalation containing from about 0.01 percent to about 5.0 percent by weight of the compound.

36. The method of claim 35, wherein the inhalation comprises administering a nasal aerosol, aerosol for inhalations, or dry powder for inhalation containing from about 0.1 percent to about 1.0 percent by weight of the compound.

37. The method of claim 26, wherein the compound is a retinoid antagonists or a alkali metal salt, alkaline earth metal salt, benzyl ester, lower alkyl ester, or 9-fluorenylmethyl ester thereof.

=> d clm 10

L22 ANSWER 10 OF 11 USPATFULL
CLM What is claimed is:

1. A substantially pure, homogeneous and isolated low affinity human Interleukin-12 receptor protein comprising an amino acid sequence selected from SEQ ID NO:2 or SEQ ID NO:3 and which binds specifically to Interleukin-12.

2. The Interleukin-12 receptor protein of claim 1 having the amino acid sequence SEQ ID NO:2.

3. The low affinity Interleukin-12 receptor protein of claim 1 wherein the Interleukin-12 receptor protein has a K.sub.D of about 2 to about 10 nM.

4. The low affinity Interleukin-12 receptor protein of claim 3 wherein the Interleukin-12 receptor protein has a K.sub.D of about 2 to about 5 nM.

5. The low affinity Interleukin-12 receptor protein of claim 4 wherein the Interleukin-12 receptor protein has the amino acid sequence SEQ ID NO:2.

6. The low affinity Interleukin-12 receptor protein of claim 4 wherein the Interleukin-12 receptor protein has the amino acid sequence SEQ ID NO:3.

7. The Interleukin-12 receptor protein of claim 1 having the amino acid sequence SEQ ID NO:3.

8. A pharmaceutical composition comprising a substantially pure, homogeneous and isolated low affinity human Interleukin-12 receptor protein comprising an amino acid sequence selected from SEQ ID NO:2 or SEQ ID NO:3 and which binds specifically to Interleukin-12 and a suitable diluent or carrier.

9. The pharmaceutical composition of claim 8 wherein the human low affinity Interleukin-12 receptor protein has the amino acid sequence SEQ ID NO:2.

10. The pharmaceutical composition of claim 8 wherein the human low affinity Interleukin-12 receptor protein has the amino acid sequence SEQ ID NO:3.

```
=> d his

     (FILE 'HOME' ENTERED AT 11:40:44 ON 11 JUL 2002)

     FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABA, WPIDS, JAPIO, BIOTECHDS,
     LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:42:50 ON 11 JUL 2002
                E LEONARD JOHN P/AU
L1          108 S E3-E5
                E LEONARD J P/AU
L2          359 S E3-E4
                E GOLDMAN SAMUEL/AU
L3           78 S E2-E9
                E GOLDMAN S/AU
L4         1458 S E3
                E OHARA RICHARD/AU
                E OHARA R/AU
L5           70 S E3
L6         2059 S L1-L5
L7          109 S L6 AND (IL-12 OR RA OR ARTHRITIS)
L8           43 S L7 AND (ANTIBOD? OR ANTAGONIST?)
L9           20 DUP REM L8 (23 DUPLICATES REMOVED)
L10           1 S L9 AND P40

     FILE 'STNGUIDE' ENTERED AT 11:51:09 ON 11 JUL 2002
L11           0 S L8 AND (FRAGMENT? OR EPITOP?)
L12           2 S RA OR RHEUMATOID ARTHRITIS

     FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABA, WPIDS, JAPIO, BIOTECHDS,
     LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:55:50 ON 11 JUL 2002
L13           3 S L9 AND (FRAGMENT? OR EPITOP?)
L14      656173 S RA OR RHEUMATOID ARTHRITIS
L15        1079 S L14 AND IL-12
L16         724 S L15 AND (ANTIBOD? OR EPITOP? OR FRAGMENT? OR ANTAGONIST)
L17          95 S L16 AND IL-12 (5A) ANTIBOD?
L18          82 DUP REM L17 (13 DUPLICATES REMOVED)
L19          18 S L18 AND P40
L20          52 S L16 AND IL-12 (5A) ANTAGONIST?
L21          44 DUP REM L20 (8 DUPLICATES REMOVED)
L22          11 S L21 AND P40

=> s l16 and p40
L23          67 L16 AND P40

=> s l23 and (antibod? or antagonist?)
L24          67 L23 AND (ANTIBOD? OR ANTAGONIST?)

=> dup rem l24
PROCESSING COMPLETED FOR L24
L25          55 DUP REM L24 (12 DUPLICATES REMOVED)

=> s l25 and (antibod? (5a) IL-12 or IL-12 (5a) antagonist?)
   8 FILES SEARCHED...
L26          21 L25 AND (ANTIBOD? (5A) IL-12 OR IL-12 (5A) ANTAGONIST?)

=> d bib ab 1-21

L26  ANSWER 1 OF 21  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN   2001:290669  BIOSIS
DN   PREV200100290669
TI   Antigen-specific T cell-mediated gene therapy in collagen-induced
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arthritis.
AU Nakajima, Atsuo; Seroogy, Christine M.; Sandora, Matthew R.; Tarner, Ingo H.; Costa, Gina L.; Taylor-Edwards, Cariel; Bachmann, Michael H.; Contag, Christopher H.; Fathman, C. Garrison (1)
CS (1) Department of Medicine, Division of Immunology and Rheumatology, School of Medicine, Stanford University, CCSR Building, Room 2225, Stanford, CA, 94305-5111: cfathman@leland.stanford.edu USA
SO Journal of Clinical Investigation, (May, 2001) Vol. 107, No. 10, pp. 1293-1301. print.
ISSN: 0021-9738.
DT Article
LA English
SL English
AB Autoantigen-specific T cells have tissue-specific homing properties, suggesting that these cells may be ideal vehicles for the local delivery of immunoregulatory molecules. We tested this hypothesis by using type II collagen-specific (CII-specific) CD4+ T hybridomas or primary CD4+ T cells after gene transfer, as vehicles to deliver an immunoregulatory protein for the treatment of collagen-induced arthritis (CIA), a mouse model of **rheumatoid arthritis** (**RA**). CII-specific T cells or hybridomas were transduced using retroviral vectors to constitutively express the **IL-12 antagonist, IL-12 p40**. Transfer of engineered CD4+ T cells after immunization significantly inhibited the development of CIA, while cells transduced with vector control had no effect. The beneficial effect on CIA of **IL-12 p40**-transduced T cells required TCR specificity against CII, since transfer of T cells specific for another antigen producing equivalent amounts of **IL-12 p40** had no effect. In vivo cell detection using bioluminescent labels and RT-PCR showed that transferred CII-reactive T-cell hybridomas accumulated in inflamed joints in mice with CIA. These results indicate that the local delivery of **IL-12 p40** by T cells inhibited CIA by suppressing autoimmune responses at the site of inflammation. Modifying antigen-specific T cells by retroviral transduction for local expression of immunoregulatory proteins thus offers a promising strategy for treating **RA**.

L26 ANSWER 2 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:518318 BIOSIS
DN PREV199900518318
TI Differential regulation of rheumatoid synovial cell interleukin-12 production by tumor necrosis factor alpha and CD40 signals.
AU Kitagawa, Minetake (1); Mitsui, Hiroshi; Nakamura, Hiroshi; Yoshino, Shinichi; Miyakawa, Shunpei; Ochiai, Naoyuki; Onobori, Makoto; Suzuki, Hiroshi; Sumida, Takayuki
CS (1) Division of Rheumatology, Department of Internal Medicine, Institute of Clinical Medicine, University of Tsukuba, 1-1-1 Tennodai, Tsukuba-shi, Ibaraki, 305-8575 Japan
SO Arthritis & Rheumatism, (Sept., 1999) Vol. 42, No. 9, pp. 1917-1926.
ISSN: 0004-3591.
DT Article
LA English
SL English
AB Objective: To investigate the roles of tumor necrosis factor alpha (TNFalpha) and the CD40-CD154 interaction in interleukin-12 (**IL-12**) production by rheumatoid synovial cells (SC). Methods: Levels of **IL-12** (**p40** and p70) in synovial tissue and culture supernatants of SC from patients with **rheumatoid arthritis** (**RA**), osteoarthritis (OA), and ankylosing spondylitis (AS) were assayed by enzyme-linked immunosorbent assay. Effects of anti-CD154 and anti-TNFalpha **antibody** on spontaneous and lipopolysaccharide (LPS)-stimulated **IL-12**

production by SC were examined. Effects of immobilized anti-CD3 treatment and depletion of CD4+ T cells on **IL-12** production were also tested. CD154 expression by synovial T cells and intracellular **IL-12** production during culture were analyzed by flow cytometry. Results: **IL-12 p40** and p70 levels in **RA** synovial tissue and spontaneous **IL-12 p40** production by SC from **RA** patients were significantly higher than the levels in OA and AS patients. Spontaneous **IL-12** production by SC from **RA** patients significantly decreased after depletion of CD4+ T cells from SC or after application of anti-CD154 **antibody**, but not by treatment with anti-TNFalpha **antibody**. Anti-CD3 **antibody** stimulation increased spontaneous **IL-12 p40** production and CD154 expression by synovial T cells. The increment of **IL-12 p40** production by anti-CD3 was abrogated by anti-CD154 **antibody**. **IL-12 p40** production was also increased by LPS stimulation. LPS-stimulated **IL-12** production was inhibited by anti-TNFalpha **antibody**, but not by T cell depletion and anti-CD154 **antibody** treatment. The TNFalpha inhibitor rolipram inhibited LPS-stimulated **IL-12 p40** production by **RA** SC more strongly than spontaneous production. TNFalpha restored LPS-stimulated **IL-12** production that had been inhibited by rolipram. Conclusion: **IL-12** production in **RA** is regulated by 2 different pathways. One pathway is T cell dependent, predominantly through a CD40-CD154 interaction, while the other is T cell independent, mediated through TNFalpha. Inhibition of **IL-12** production by interference with CD40-CD154 interaction and TNFalpha production may be a potential therapeutic strategy for treating **RA**.

L26 ANSWER 3 OF 21 WPIDS (C) 2002 THOMSON DERWENT
AN 2001-244560 [25] WPIDS
DNC C2001-073385
TI Composition comprising interleukin-12 **p40** and IL-B30 polypeptide or its segment, useful for ameliorating **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor.
DC B04 D16
IN DE WAAL MALEFYT, R; KASTELEIN, R A; LIRA, S A; NARULA, S K; OPPMANN, B; RENNICK, D M; WIEKOWSKI, M T
PA (SCHE) SCHERING CORP
CYC 92
PI WO 2001018051 A2 20010315 (200125)* EN 69p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CZ DE DK DM DZ EE ES FI GB GD GE HR HU ID IL IN IS JP KG KR KZ LC LK LR LT LU LV MA MD MG MK MN MX MZ NO NZ PL PT RO RU SE SG SI SK SL TJ TM TR TT TZ UA UZ VN YU ZA
AU 2000073608 A 20010410 (200137)
EP 1210434 A2 20020605 (200238) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
ADT WO 2001018051 A2 WO 2000-US24686 20000908; AU 2000073608 A AU 2000-73608 20000908; EP 1210434 A2 EP 2000-961688 20000908, WO 2000-US24686 20000908
FDT AU 2000073608 A Based on WO 200118051; EP 1210434 A2 Based on WO 200118051
PRAI US 1999-164616P 19991110; US 1999-393090 19990909
AB WO 200118051 A UPAB: 20010508
NOVELTY - A composition (I) comprising a substantially pure polypeptide comprising a number of distinct segments of at least 7 contiguous amino acids from interleukin (**IL**)-**12 p40** and/or IL-B30, and a substantially pure polypeptide comprising a segment of at

least 11 contiguous amino acids from **IL-12 p40** and/or IL-B30.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated or recombinant nucleic acid (II) encoding (I);

(2) a cell (III) comprising (II);

(3) a nucleic acid (IV) which hybridizes under wash conditions of 30 minutes at 50 deg. C and less than 1M salt to the natural mature coding portion of primate **IL-12 p40** and IL-B30;

(4) an **antagonist** (V) of **IL-12 p40**/IL-B30 combined with a tumor necrosis factor-alpha (TNF alpha) **antagonist**, an **IL-12 antagonist**, IL-10, or steroids;

(5) a binding compound (VI) comprising an antigen binding site from an **antibody**, which specifically binds to (I) and comprising a substantially pure polypeptide comprising **IL-12 p40** and IL-B30 polypeptide, or a polypeptide comprising **IL-12 p40** fused to IL-B30, but not to either **IL-12 p40** or IL-B30 polypeptide;

(6) a kit (VII) comprising:

(a) (I), and a compartment comprising the polypeptide, or instructions for use or disposal of reagents in the kit;

(b) (II), and a compartment comprising (II), a compartment further comprising a primate **IL-12 p40** or IL-B30, or instructions for use or disposal of reagents in the kit or (VI); and

(c) a compartment comprising (VI), or instructions for use or disposal of reagents in the kit;

(7) producing (M1) an antigen:**antibody** complex, involves contacting, under appropriate conditions, a primate **IL-12 p40**/IL-B30 composition with (VI), allowing the complex to form;

(8) a composition (VIII) comprising (VI) which is sterile, or (VI) and a carrier such as an aqueous compound, including water, saline, and/or buffer;

(9) increasing (M2) the secretion of a primate IL-B30, involves expressing the polypeptide with **IL-12 p40** or increasing the secretion of a primate **IL-12 p40** involves expressing the **IL-12 p40** with IL-B30; and

(10) screening (M3) for a receptor which binds (I) involves contacting the complex to a cell expressing the receptor under conditions allowing the complex to bind to the receptor, forming a detectable interaction.

ACTIVITY - Antirheumatic; antiarthritic; osteopathic; antiarthritic; neuroprotective; antiarteriosclerotic; cerebroprotective; vasotropic; cytostatic; antitumor; immunosuppressive.

MECHANISM OF ACTION - Modulator of physiology or development of cell in host; inducer of memory T-cell proliferation (claimed); modulator of trafficking or activation of leukocyte.

No supporting data is given.

USE - (I) is useful for modulating physiology or development of a cell or tissue in a host organism by contacting the cell with (I) or (V), resulting in an increased or decreased production of Interferon-gamma (IFN gamma), an enhanced Th1 response such as anti-tumor effect, adjuvant effect, anti-viral effect or antagonized allergic effect, and amelioration of an autoimmune condition or a chronic inflammatory condition. The contacting is in combination with IL-18, **IL-12**, radiation therapy or chemotherapy, an immune adjuvant or an anti-viral therapeutic. The **antagonist** is an **antibody** against **IL-12** receptor subunit beta 1. The **antagonist** or agonist of mammalian IL-B30 protein is useful for modulating the inflammatory response in an animal, by contacting cells in the animal with

the agonist or **antagonist**, where the animal exhibits signs or symptoms of an acute phase inflammatory response in skin, lung, gastrointestinal, or liver tissue. The modulation is accelerating maturation of neutrophils into platelets and has an effect on immunoglobin A and G (IgA and IgG) . The **antagonist** is an **antibody** which binds to the mammalian IL-B30 or blocks signaling mediated by mammalian IL-B30. The **antagonist** or agonist is administered in combination with an anti-inflammatory cytokine agonist or **antagonist**, an analgesic, an anti-inflammatory agent, or a steriod. IL-B30 or its agonist is useful inducing the proliferation of memory T-cells (all claimed).

Agonist or **antagonist** of IL-B30 protein is useful for modulating the trafficking or activation of a leukocyte in an animal experiencing science or symptoms of autoimmunity, an inflammatory condition, tissue specific autoimmunity, degenerative autoimmunity, **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis, delayed hypersensitivities, skin grafting, a transplant, spinal injury, stroke, neurodegeneration, an infectious disease, ischemia, cancer, tumors, multiple myeloma, Castleman's disease, postmenopausal osteoporosis or IL-6-associated diseases.

IL-12 p40/IL-B30 is useful as an immunogen for the production a antisera or **antibodies** specific for binding. (I) is useful for in vitro assays, scientific research, and the synthesis or manufacture of nucleic acids or **antibodies**. (II) is useful in forensic science.

Dwg.0/0

L26 ANSWER 4 OF 21 WPIDS (C) 2002 THOMSON DERWENT
AN 1999-458684 [38] WPIDS
DNC C1999-134705
TI New **antibodies** to human interleukin-12, used for treating diseases associated with increased **IL-12** bioactivity such as autoimmune disorders, e.g. multiple sclerosis.
DC B04 D16
IN GATELY, M K; PRESKY, D H; GATELY, M
PA (HOFF) HOFFMANN LA ROCHE & CO AG F; (HOFF) HOFFMANN LA ROCHE INC
CYC 85
PI WO 9937682 A2 19990729 (199938)* EN 46p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZW
ZA 9900452 A 19990929 (199947) 48p
AU 9925177 A 19990809 (200001)
BR 9907743 A 20001017 (200056)
EP 1049717 A2 20001108 (200062) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU NL PT SE
US 6225117 B1 20010501 (200126)
CN 1288468 A 20010321 (200137)
KR 2001034315 A 20010425 (200164)
MX 2000007124 A1 20010301 (200170)
JP 2002501085 W 20020115 (200207) 50p
ADT WO 9937682 A2 WO 1999-EP202 19990115; ZA 9900452 A ZA 1999-452 19990121; AU 9925177 A AU 1999-25177 19990115; BR 9907743 A BR 1999-7743 19990115, WO 1999-EP202 19990115; EP 1049717 A2 EP 1999-904780 19990115, WO 1999-EP202 19990115; US 6225117 B1 Provisional US 1998-72333P 19980123, US 1999-232522 19990119; CN 1288468 A CN 1999-802310 19990115; KR 2001034315 A KR 2000-708036 20000722; MX 2000007124 A1 MX 2000-7124 20000720; JP 2002501085 W WO 1999-EP202 19990115, JP 2000-528602 19990115
FDT AU 9925177 A Based on WO 9937682; BR 9907743 A Based on WO 9937682; EP

1049717 A2 Based on WO 9937682; JP 2002501085 W Based on WO 9937682
PRAI US 1998-72333P 19980123; US 1999-232522 19990119
AB WO 9937682 A UPAB: 19991122
NOVELTY - New **antibodies** to human interleukin-12 are produced using a mammal which is deficient in the gene encoding the p35 or **p40** subunit of **IL-12**.

DETAILED DESCRIPTION - (A) An **antibody** to the human interleukin (**IL**)-**12** p75 heterodimer which consists of a p35 subunit and a **p40** subunit, where the **antibody**:

(i) immunologically reacts with an **epitope** presented by the p75 heterodimer of human **IL-12**, but is not immunologically reactive with an **epitope** presented by the **p40** subunit; and

(ii) is produced from a mammal, preferably a mouse which is deficient in the gene encoding the p35 subunit or the **p40** subunit of **IL-12**.

INDEPENDENT CLAIMS are also included for the following:

(1) a monoclonal **antibody** (MAb) to human **IL-12** which consists of a p35 subunit and a **p40** subunit forming a p75 heterodimer, where the MAb;

(i) immunologically reacts with an **epitope** presented by the p75 heterodimer of human **IL-12**, but is not immunologically reactive with any **epitope** presented by the **p40** subunit; and

(ii) neutralizes at least 90% of the bioactivity of human **IL-12**;

(2) a hybridoma that produces an **antibody** as in (A) or (1).

ACTIVITY - The **antibodies** can neutralize **IL-12** bioactivity as determined by ability to block **IL-12** stimulated phytohemagglutinin A (PHA)-activated lymphoblast proliferation and interferon- gamma production by PHA-activated lymphoblasts. The 5F2, 16F2, 16G2 and 20E11 **antibodies** were able to inhibit human **IL-12** stimulated PHA activated human lymphoblast proliferation by at least 90%. These anti-human heterodimer specific **IL-12 antibodies** were able to inhibit greater than 90% of **IL-12** stimulated IFN-gamma production when used at 0.5 micro g/ml.

USE - The **antibodies** can be used for controlling diseases with pathologies that are mediated through immune mechanisms, particularly diseases associated with increased **IL-12** bioactivity that results in aberrant Th1-type helper cell activity like autoimmune disorders, e.g. multiple sclerosis, **rheumatoid arthritis**, autoimmune diabetes mellitus, and inflammatory bowel disease (IBD) including Crohn's disease and ulcerative colitis (claimed). They can also be used to treat transplantation/graft-versus-host disease and septic shock.

ADVANTAGE - The anti-**IL-12 antibodies** exhibit higher potency and greater efficacy than known heterodimer specific **IL-12 antibodies**.

Dwg.0/7

L26 ANSWER 5 OF 21 BIOTECHDS COPYRIGHT 2002 THOMSON DERWENT AND ISI
AN 2001-08257 BIOTECHDS
TI Composition containing interleukin-12 **p40** and IL-B30 protein or its segment, useful for ameliorating **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor;
vector-mediated gene transfer and expression in host cell, **antibody** and **antagonist**
AU Oppmann B; De Waal Malefyt R; Rennick D M; Kastelein R A; Wiekowski M T; Lira S A; Narula S K
PA Schering-USA

LO Kenilworth, NJ, USA.
PI WO 2001018051 15 Mar 2001
AI WO 2000-US24686 8 Sep 2000
PRAI US 1999-164616 10 Nov 1999; US 1999-393090 9 Sep 1999
DT Patent
LA English
OS WPI: 2001-244560 [25]
AB A composition containing a substantially pure protein containing a number of distinct segments of at least 7 contiguous amino acids from interleukin (**IL**)-**12 p40** and/or IL-B30, and a substantially pure protein containing a segment of at least 11 contiguous amino acids from **IL-12 p40** and/or IL-B30, is new. Also claimed are: a recombinant nucleic acid encoding the protein; a cell containing the nucleic acid; a nucleic acid which hybridizes under wash conditions of 30 min at 50 deg and less than 1M salt to the natural mature coding portion of primate **IL-12 p40** and IL-B30; an **antagonist** of **IL-12 p40**/IL-B30 combined with a tumor necrosis factor-alpha (TNF-alpha) **antagonist**, an **IL-12 antagonist**, IL-10 or steroids; a binding compound containing an antigen binding site from an **antibody** which specifically binds to the protein; a kit containing the composition, polynucleotide and a binding compound; producing an antigen: **antibody** complex; a composition containing a binding compound; increasing the secretion of a primate IL-B30; and screening for a receptor which binds the composition. The composition is useful for modulating physiology or development of a cell or tissue0. (69pp)

L26 ANSWER 6 OF 21 USPATFULL
AN 2002:157653 USPATFULL
TI Triazine compounds
IN Ono, M, Lexington, MA, UNITED STATES
Sun, Lijun, Harvard, MA, UNITED STATES
Zhang, Shijie, Nashua, NH, UNITED STATES
Przewloka, Teresa, Burlington, MA, UNITED STATES
James, David A., Cambridge, MA, UNITED STATES
Ding, Wenli, Worcester, MA, UNITED STATES
Wada, Yumiko, Waltham, MA, UNITED STATES
PI US 2002082259 A1 20020627
AI US 2001-6624 A1 20011130 (10)
RLI Continuation-in-part of Ser. No. US 2000-594362, filed on 15 Jun 2000, PENDING
DT Utility
FS APPLICATION
LREP Y. ROCKY TSAO, Fish & Richardson P.C., 225 Franklin Street, Boston, MA, 02110-2804
CLMN Number of Claims: 48
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 879
AB This invention relates to triazine compounds of formula (I): ##STR1##

R.sub.1 is , aryl, ##STR2##

or heteroaryl; each of R.sub.2, R.sub.4, and R.sub.5, independently, is R.sup.c, halogen, nitro, nitroso, cyano, azide, isothionitro, SR.sup.c, or OR.sup.c; R.sub.3 is R.sup.c, alkenyl, alkynyl, aryl, heteroaryl, cyclyl, heterocyclyl, OR.sup.c, OC(O)R.sup.c, SO.sub.2R.sup.c, S(O)R.sup.c, S(O.sub.2)NR.sup.cR.sup.d, SR.sup.c, NR.sup.cR.sup.d, NR.sup.cCOR.sup.d, NR.sup.cC(O)OR.sup.d, NR.sup.cC(O)NR.sup.cR.sup.d, NR.sup.cSO.sub.2R.sup.d, COR.sup.c, C(O)OR.sup.c, or C(O)NR.sup.cR.sup.d; n is 0, 1, 2, 3, 4, 5, 6, or 7; X is O, S, S(O),

S(O.sub.2), or NR.sup.c; Y is a covalent bond, CH.sub.2, C(O), C.dbd.N--R.sup.c, C.dbd.N--OR.sup.c, C.dbd.N--SR.sup.c, O, S, S(O), or S(O.sub.2); Z is N; and W is O, S, S(O), S(O.sub.2), NR.sup.c, or NC(O)R.sup.c; in which each of R.sup.a and R.sup.b, independently, is H, alkyl, aryl, heteroaryl; and each of R.sup.c and R.sup.d, independently, is H, alkyl, or alkylcarbonyl.

L26 ANSWER 7 OF 21 USPATFULL
AN 2002:84902 USPATFULL
TI Nucleic acids, proteins and **antibodies**
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PI US 2002044941 A1 20020418
AI US 2001-925302 A1 20010810 (9)
RLI Continuation-in-part of Ser. No. WO 2000-US5918, filed on 8 Mar 2000, UNKNOWN
PRAI US 1999-124270P 19990312 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 21121
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel lung cancer related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "lung cancer antigens," and **antibodies** that immunospecifically bind these polypeptides, and the use of such lung cancer polynucleotides, antigens, and **antibodies** for detecting, treating, preventing and/or prognosing disorders of the lung, including, but not limited to, the presence of lung cancer and lung cancer metastases. More specifically, isolated lung cancer nucleic acid molecules are provided encoding novel lung cancer polypeptides. Novel lung cancer polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human lung cancer polynucleotides, polypeptides, and/or **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the lung, including lung cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and **antagonists** of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

L26 ANSWER 8 OF 21 USPATFULL
AN 2001:229210 USPATFULL
TI Methods for enhancing oral tolerance and treating autoimmune disease using inhibitors of interleukin-12
IN Strober, Warren, Bethesda, MD, United States
Kelsall, Brian, Washington, DC, United States
Marth, Thomas, Kensington, MD, United States
PA Government of the United States of America, Department of Health and Human Services (U.S. corporation)
PI US 2001051159 A1 20011213
AI US 2000-732502 A1 20001207 (9)
RLI Continuation of Ser. No. US 1999-284169, filed on 9 Apr 1999, ABANDONED A 371 of International Ser. No. WO 1996-US16007, filed on 11 Oct 1996, UNKNOWN
DT Utility

FS APPLICATION
LREP mary 1. miller THE CANDLER BUILDING, needle & rosenberg, p.c., 127 peachtree street, n.e., atlanta, GA, 30303-1811
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1252
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a method for enhancing oral tolerance to an antigen associated with an autoimmune disease in a subject having the autoimmune disease comprising orally administering to the subject an antigen associated with the autoimmune disease and administering an inhibitor of interleukin-12 in amounts sufficient to enhance oral tolerance. Also provided in the present invention is a method for treating or preventing an autoimmune disease in a subject comprising orally administering to the subject an antigen associated with the autoimmune disease and administering an inhibitor of interleukin-12 in amounts sufficient to treat or prevent the autoimmune disease, thereby treating or preventing the autoimmune disease.

L26 ANSWER 9 OF 21 USPATFULL
AN 2001:221075 USPATFULL
TI Retinoid **antagonists** and use thereof
IN Bollag, Werner, Basel, Switzerland
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of
Mohr, Peter, Basel, Switzerland
Panina-Bordignon, Paola, Milan, Italy
Rosenberger, Michael, Caldwell, NJ, United States
Sinigaglia, Francesco, Milan, Italy
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6326397 B1 20011204
AI US 1999-307009 19990507 (9)
RLI Continuation-in-part of Ser. No. US 1998-189189, filed on 10 Nov 1998
DT Utility
FS GRANTED
EXNAM Primary Examiner: Killos, Paul J.
LREP Johnston, George W., Parise, John P.
CLMN Number of Claims: 16
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 1573
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel retinoid **antagonists** of the formula I ##STR1##

wherein the dotted bond can be either hydrogenated or form a double bond; and, when the dotted bond forms a double bond, R.sup.1 is lower alkyl and R.sup.2 is hydrogen; and, when the dotted bond is hydrogenated, R.sup.1 and R.sup.2 taken together are methylene to form a cis-substituted cyclopropyl ring; R.sup.3 is hydroxy or lower alkoxy; R.sup.4 is alkyl or alkoxy; and R.sup.5 and R.sup.6 are, independently, a C.sub.4-12 alkyl or a 5-12 cycloalkyl substituent containing from 1-3 rings which are either unsubstituted or substituted with from 1-3 lower alkyl groups, with the carbon atom of R.sup.5 and R.sup.6 being linked to the remainder of the molecule to form a quaternary carbon atom pharmaceutically acceptable salts of carbocylic acids of the formula I; as well as method for the treatment of osteoporosis and preneoplastic and neoplastic diseases, and a method for reducing or abolishing adverse events in subjects receiving retinoid agonist treatment by administering a retinoid **antagonist**.

L26 ANSWER 10 OF 21 USPATFULL

AN 2001:63494 USPATFULL
TI **Antibodies** against human **IL-12**
IN Gately, Maurice Kent, Parsippany, NJ, United States
Presky, David Howard, Glen Ridge, NJ, United States
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6225117 B1 20010501
AI US 1999-232522 19990119 (9)
PRAI US 1998-72333P 19980123 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: DiBrino, Marianne
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1122
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel p75 heterodimer specific anti-human **IL-12 antibodies** that are characterized by a higher potency and greater efficacy in neutralizing human **IL-12** bioactivity than known heterodimer specific **IL-12** monoclonal **antibodies**. The heterodimer specific **antibodies** recognize one or more **epitopes** of the human **IL-12** p75 heterodimer, but do not bind to the **p40** subunit alone. The heterodimer specific **IL-12 antibodies** neutralize rhesus monkey **IL-12** bioactivity with a potency similar to their potency for neutralizing human **IL-12** bioactivity making them useful **IL-12 antagonists** for in vivo studies in the rhesus monkey.

L26 ANSWER 11 OF 21 USPATFULL
AN 2000:138395 USPATFULL
TI Treatment of T-helper cell type 2-mediated immune disease by retinoid **antagonists**
IN Bollag, Werner, Basel, Switzerland
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of
Panina-Bordignon, Paola, Milan, Italy
Sinigaglia, Francesco, Milan, Italy
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 6133309 20001017
AI US 1998-189189 19981110 (9)
PRAI EP 1997-119776 19971112
DT Utility
FS Granted
EXNAM Primary Examiner: Travers, Russell
LREP Johnston, George W., Epstein, William H., Parise, John P.
CLMN Number of Claims: 37
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 780
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Retinoids with retinoid receptor **antagonistic** activity, pharmaceutically acceptable salts and pharmaceutically acceptable hydrolyzable esters thereof, have been found efficacious in treating T-helper cell type 2 (Th2)-mediated immune diseases, such as immunoglobulin E (IgE)-mediated allergic diseases.

L26 ANSWER 12 OF 21 USPATFULL
AN 2000:98551 USPATFULL
TI Treatment of papillomavirus-associated lesions

IN Stanley, Margaret Anne, Cambridge, United Kingdom
Scarpini, Cinzia Giuseppina, Cambridge, United Kingdom
PA Cambridge University Technical Services, Ltd., Cambridge, United Kingdom (non-U.S. corporation)
PI US 6096869 20000801
AI US 1996-621841 19960322 (8)
PRAI GB 1995-5784 19950322
DT Utility
FS Granted
EXNAM Primary Examiner: Park, Hankyel
LREP Klarquist Sparkman Campbell Leigh & Whinston, LLP
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1293
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Interleukin-12 (**IL-12**) or a functional analogue thereof, or a polynucleotide encoding **IL-12** or encoding a functional analogue thereof, is used as a therapeutic material or adjuvant in treating papillomavirus-associated lesions e.g. warts due to HPV 6 and/or 11, e.g. condyloma acuminata. **IL-12** or a vector encoding it for endogenous production can be used together with a vaccine such as a papillomavirus antigen, or a vector encoding a papillomavirus antigen.

L26 ANSWER 13 OF 21 USPATFULL
AN 2000:87729 USPATFULL
TI Method of converting a Th2-type allergic immune response into a Th1-type immune response
IN DeKruyff, Rosemarie H., Stanford, CA, United States
Umetsu, Dale T., Stanford, CA, United States
PA The Board of Trustees of the Leland Stanford Junior University, Palo Alto, CA, United States (U.S. corporation)
PI US 6086898 20000711
AI US 1999-339068 19990623 (9)
PRAI US 1998-90390P 19980623 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: Ewoldt, Gerald R.
LREP Bozicevic, Field & Francis, Sherwood, Pamela
CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN 17 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 1767
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods are provided for the treatment of allergic and other immune disorders associated with overproduction of Th2 type cytokines by antigen specific T cells. Immunotherapy with adjuvants, as provided in the present invention, greatly inhibits the development of airway hyperreactivity and airway inflammation. Such immunotherapy is shown to reverse ongoing airway disease, and convert allergic inflammatory responses into protective immune responses. Conditions of particular interest include allergic conditions associated with production of Th2 cytokines and/or IgE **antibodies**, asthma, allergic rhinitis, and anaphylactic reactions. The addition of adjuvant induces a Th1-type immune response and can redirect an established Th2-type response to a Th1-type response for the selected antigen. Preferably, antigen-specific IgE production is reduced without altering the intensity of the antigen-specific proliferative response. One particularly preferred adjuvant for use in accordance with the present invention is a Listeria adjuvant.

L26 ANSWER 14 OF 21 USPATFULL
AN 2000:87707 USPATFULL
TI Methods and compositions for the inhibition of interleukin-12 production
IN Karp, Christopher L., Lutherville, MD, United States
Trinchieri, Giorgio, Wynnewood, PA, United States
Wysocka, Maria, Wynnewood, PA, United States
Griffin, Diane E., Hunt Valley, MD, United States
PA The Wistar Insitute, Philadelphia, PA, United States (U.S. corporation)
Johns Hopkins University, Baltimore, MD, United States (U.S. corporation)
PI US 6086876 20000711
AI US 1998-19862 19980206 (9)
PRAI US 1997-37722P 19970207 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Kemmerer, Elizabeth; Assistant Examiner: Romeo, David S.
LREP Akin, Gump, Strauss, Hauer & Feld, L.L.P.
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 13 Drawing Figure(s); 18 Drawing Page(s)
LN.CNT 1487
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention includes compositions and methods for selective suppression of **IL-12** production in a cell. Methods of treating a human having a disease associated with dysregulated **IL-12** production are also provided.

L26 ANSWER 15 OF 21 USPATFULL
AN 2000:50737 USPATFULL
TI Methods and compositions for modulating responsiveness to corticosteroids
IN Sekut, Les, Westborough, MA, United States
Carter, Adam, Newburyport, MA, United States
Ghayur, Tariq, Grafton, MA, United States
Banerjee, Subhashis, Shrewsbury, MA, United States
Tracey, Daniel E., Harvard, MA, United States
PA BASF Aktiengesellschaft, Rheinland Pfalz, Germany, Federal Republic of (non-U.S. corporation)
PI US 6054487 20000425
AI US 1997-820692 19970318 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Jarvis, William R. A.
LREP Lahive & Cockfield, LLP
CLMN Number of Claims: 46
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 2404
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Method for modulating responsiveness to corticosteroids in a subject are provided. In the method of the invention, an agent which antagonizes a factor that regulates production of IFN-.gamma. in the subject is administered to the subject in combination with a corticosteroid such that responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject. In one embodiment, the agent is an interferon-.gamma. inducing factor (IGIF) **antagonist**. In another embodiment, the agent is an interleukin-12 (**IL-12**) **antagonist**. In a preferred embodiment, the agent is an inhibitor of a caspase family protease, preferably an ICE inhibitor. In another preferred embodiment,

the agent is an anti-**IL-12** monoclonal **antibody**. Other preferred agents include phosphodiesterase IV inhibitors and beta-2 agonists. The methods of the invention can be used in the treatment of a variety of inflammatory and immunological diseases and disorders. Pharmaceutical compositions comprising an agent which antagonizes a factor that regulates production of IFN-.gamma. in a subject, a corticosteroid and a pharmaceutically acceptable carrier are also provided. A preferred composition comprises an ICE inhibitor, a corticosteroid and a pharmaceutically acceptable carrier.

L26 ANSWER 16 OF 21 USPATFULL
AN 1999:155952 USPATFULL
TI Dihomo-seco-cholestanes
IN Barbier, Pierre, Rixheim, France
Mohr, Peter, Basel, Switzerland
Muller, Marc, Saint-Louis, France
Self, Christopher, West Caldwell, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5994569 19991130
AI US 1998-115188 19980714 (9)
PRAI EP 1997-112225 19970717
DT Utility
FS Granted
EXNAM Primary Examiner: Dees, Jose' G.; Assistant Examiner: Badio, Barbara
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 28
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1220
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Polyunsaturated 24a,24b-dihomo-9,10-secocholestane derivatives of formula ##STR1## wherein A is a single or double bond,

B.sup.1 and B.sup.2 are each independently CH.dbd.CH or C.tbd.C,

T is CH.sub.2 or CH.sub.2 CH.sub.2,

X is --CH.sub.2 -- or >C.dbd.CH.sub.2,

R.sup.1 is H, F or OH,

R.sup.2 and R.sup.3 are each independently lower alkyl or CF.sub.3, or

C(R.sup.2,R.sup.3) is C.sub.3-6 -cycloalkyl,

are useful in the treatment or prevention of vitamin D dependent disorders and of **IL-12**-dependent autoimmune diseases, particularly psoriasis, basal cell carcinomas, disorders of keratinization and keratosis, leukemia, osteoporosis, hyperparathyroidism accompanying renal failure, multiple sclerosis, transplant rejection, graft vs. host disease, **rheumatoid arthritis**, insulin-dependent diabetes mellitus, inflammatory bowel disease, septic shock and allergic encephalomyelitis.

L26 ANSWER 17 OF 21 USPATFULL
AN 1999:75759 USPATFULL
TI Low affinity human **IL-12** beta2 receptor
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States
Presky, David Howard, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5919903 19990706
AI US 1997-914520 19970819 (8)

RLI Division of Ser. No. US 1996-685118, filed on 23 Jul 1996
PRAI US 1995-1701P 19950801 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 2
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1531
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A recombinant human **IL-12** receptor complex produced on the surface of a non-human mammalian cell and free from other human proteins, the complex comprising the beta1 receptor protein complexed with a beta2 receptor protein, which complex is capable of binding to human **IL-12** with high affinity. A recombinant human **IL-12** beta2 receptor protein produced on the surface of a non-human mammalian cell, free from other human proteins, in its active form. In addition, a non-human mammalian cell having expressed on its surface the recombinant human **IL-12** beta2 receptor protein or the recombinant human **IL-12** receptor complex, which cell proliferates in the presence of human **IL-12**. A non-human mammalian cell having the human **IL-12** beta2 receptor protein or the complex expressed on its surface and which proliferates in response to human **IL-12** is useful for determining whether a given compound inhibits biological activity of human **IL-12** or is an **IL-12** agonist.

L26 ANSWER 18 OF 21 USPATFULL
AN 1998:160106 USPATFULL
TI **Antibodies** to receptors for human interleukin-12
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States
Presky, David Howard, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5852176 19981222
AI US 1997-915495 19970820 (8)
RLI Division of Ser. No. US 1996-685118, filed on 23 Jul 1996
PRAI US 1995-1701P 19950801 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 1
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1381
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB **Antibodies** to human **IL-12** beta 2 receptor protein or an **IL-12** receptor complex, the complex comprising the beta1 receptor protein complexed with a beta2 receptor protein, which complex is capable of binding to human **IL-12** with high affinity.

L26 ANSWER 19 OF 21 USPATFULL
AN 1998:147252 USPATFULL
TI DNA encoding receptors for the beta-2 chain of human **IL-12**
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States
Presky, David Howard, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5840530 19981124

AI US 1996-685118 19960723 (8)
PRAI US 1995-1701P 19950801 (60)
US 1996-18674P 19960530 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Draper, Garnette D.
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1424
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A recombinant human **IL-12** beta2 receptor protein produced on the surface of a non-human mammalian cell, free from other human proteins, in its active form. In addition, a non-human mammalian cell having expressed on its surface the recombinant human **IL-12** beta2 receptor protein, which cell proliferates in the presence of human **IL-12**. A non-human mammalian cell having the human **IL-12** beta2 receptor protein on its surface and which proliferates in response to human **IL-12** is useful for determining whether a given compound inhibits biological activity of human **IL-12** or is an **IL-12** agonist.

L26 ANSWER 20 OF 21 USPATFULL
AN 1998:135151 USPATFULL
TI Human receptor for interleukin-12
IN Chua, Anne On, Wayne, NJ, United States
Gubler, Ulrich Andreas, Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5831007 19981103
AI US 1995-419652 19950411 (8)
RLI Division of Ser. No. US 1994-248532, filed on 31 May 1994, now patented, Pat. No. US 5536657 which is a continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John
LREP Johnston, George W., Epstein, William H., Bucholz, Briana C.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 35 Drawing Figure(s); 26 Drawing Page(s)
LN.CNT 1937
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to substantially pure Interleukin-12 receptor cDNAs and protein and uses therefore. The Interleukin-12 receptor is shown to be a member of the cytokine receptor superfamily and has a high homology to human gp130.

L26 ANSWER 21 OF 21 USPATFULL
AN 96:63048 USPATFULL
TI Recombinant DNA encoding human receptor for interleukin-12
IN Chua, Anne O., Wayne, NJ, United States
Gubler, Ulrich A., Glen Ridge, NJ, United States
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)
PI US 5536657 19960716
AI US 1994-248532 19940531 (8)
RLI Continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John

LREP Gould, George M., Johnston, George W., Kass, Alan P.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 34 Drawing Figure(s); 25 Drawing Page(s)
LN.CNT 1755
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to substantially pure Interleukin-12 receptor cDNAs and protein and uses therefore. The Interleukin-12 receptor is shown to be a member of the cytokine receptor superfamily and has a high homology to human gp130.

=> d clm 12 15 18

L26 ANSWER 12 OF 21 USPATFULL
CLM What is claimed is:

1. Pharmaceutical treatment material comprising in combination (i) **IL-12**, or a functional homologue thereof, for use as an adjuvant, and (ii) a protein consisting essentially of at least one antigenic portion of a papillomavirus protein, wherein (a) the papillomavirus is selected from the group consisting of HPV types 6, 11, 16 and 18, and (b) the papillomavirus protein is selected from the group consisting of E6, E7, L1 and L2 proteins.

2. The pharmaceutical treatment material according to claim 1, wherein the papillomavirus protein is selected from the group consisting of E7 and L2.

3. The pharmaceutical treatment material according to claim 1, wherein the vaccine adjuvant is **IL-12** or a protein that differs from **IL-12** by one or more conservative amino acid substitutions and which retains IL12 activity.

4. The pharmaceutical treatment material according to claim 1, wherein the vaccine adjuvant is **IL-12**.

5. Pharmaceutical treatment material comprising in combination (i) **IL-12**, or a functional homologue thereof, for use as an adjuvant, and (ii) a nucleic acid molecule encoding a protein consisting essentially of at least one antigenic portion of a papillomavirus protein, wherein (a) the papillomavirus is selected from the group consisting of HPV types 6, 11, 16 and 18, and (b) the papillomavirus protein is selected from the group consisting of E6, E7, L1 and L2 proteins.

6. The pharmaceutical treatment material according to claim 5, wherein the papillomavirus protein is selected from the group consisting of E7 and L2.

7. The pharmaceutical treatment material according to claim 5, wherein the vaccine adjuvant is **IL-12** or a protein that differs from **IL-12** by one or more conservative amino acid substitutions and which retains IL12 activity.

8. The pharmaceutical treatment material according to claim 5, wherein the vaccine adjuvant is **IL-12**.

9. The pharmaceutical treatment material of claim 1, wherein the papillomavirus is HPV type 16.

10. The pharmaceutical treatment material of claim 1, wherein the papillomavirus protein is selected from the group consisting of E6 and

E7.

11. The pharmaceutical treatment material of claim 1, wherein the papillomavirus protein is E7.

12. The pharmaceutical treatment material of claim 1, wherein the papillomavirus virus is HPV type 16 and the papillomavirus protein is E7.

13. The pharmaceutical treatment material of claim 5, wherein the papillomavirus virus is HPV type 16 and the papillomavirus protein is E7.

L26 ANSWER 15 OF 21 USPATFULL

CLM What is claimed is:

1. A method for modulating responsiveness to a corticosteroid in a subject, comprising administering to the subject suffering from a condition normally responsive to corticosteroid therapy: an interleukin-1 .beta. converting enzyme (ICE) inhibitor being administered at a dosage and by a route sufficient to inhibit production of IFN-.gamma. in the subject; and a corticosteroid, such that responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.

2. The method of claim 1, wherein the ICE inhibitor is an IFN-.gamma. inducing factor (IGIF) **antagonist**, the ICE inhibitor being administered at a dosage and by a route sufficient to inhibit IGIF activity in the subject.

3. The method of claim 1, wherein the corticosteroid is selected from the group consisting of cortisone, hydrocortisone, beclomethasone, flunisolide, prednisone, prednisolone, methylprednisolone, triamcinolone, deflazacort, betamethasone and dexamethasone.

4. The method of claim 1, wherein the subject is suffering from septic shock.

5. The method of claim 1, wherein the subject is suffering from Crohn's disease.

6. The method of claim 1, wherein the subject is suffering from asthma.

7. The method of claim 1, wherein the subject is suffering from graft versus host disease or transplant rejection.

8. The method of claim 1, wherein the subject is suffering from an autoimmune disease or disorder.

9. The method of claim 1, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of asthma, adult respiratory distress syndrome, systemic lupus erythematosus, inflammatory bowel disease, Crohn's disease, ulcerative colitis, multiple sclerosis, insulin-dependent diabetes mellitus, autoimmune arthritis, **rheumatoid arthritis**, juvenile **rheumatoid arthritis**, psoriatic arthritis, inflammatory pulmonary syndrome, pemphigus vulgaris, idiopathic thrombocytopenic purpura, autoimmune meningitis, myasthenia gravis, autoimmune thyroiditis, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, Sjogren's Syndrome, keratoconjunctivitis sicca secondary to Sjogren's Syndrome, alopecia areata, allergic responses due to arthropod bite reactions, aphthous

ulcer, iritis, conjunctivitis, keratoconjunctivitis, cutaneous lupus erythematosus, scleroderma, vaginitis, proctitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, erythema nodosum leprosum, autoimmune uveitis, allergic encephalomyelitis, aplastic anemia, pure red cell anemia, idiopathic thrombocytopenia, polychondritis, Wegener's granulomatosis, chronic active hepatitis, Graves ophthalmopathy, primary biliary cirrhosis, uveitis posterior and interstitial lung fibrosis.

10. The method of claim 1, wherein the subject is suffering from an acute inflammatory disorder.

11. The method of claim 1, wherein the subject is suffering from a chronic inflammatory disorder.

12. The method of claim 1, wherein the ICE inhibitor and corticosteroid are administered such that steroid resistance in the subject is reversed, as compared to when a corticosteroid alone is administered to the subject.

13. The method of claim 1, wherein the ICE inhibitor and corticosteroid are administered such that steroid sensitivity in the subject is increased, as compared to when a corticosteroid alone is administered to the subject.

14. The method of claim 1, wherein the ICE inhibitor and the corticosteroid are administered to the subject according to a schedule that reduces the dosage of the corticosteroid over time and e method ameliorates a steroid rebound effect associated with administration of reduced dosages of the corticosteroid.

15. A method for modulating responsiveness to corticosteroids in a subject, comprising administering to the subject suffering from a condition normally responsive to corticosteroid therapy, an interleukin-1 .beta. converting enzyme (ICE) inhibitor; and a corticosteroid, such that responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.

16. The method of claim 15, wherein the corticosteroid is selected from the group consisting of cortisone, hydrocortisone, beclomethasone, flunisolide, prednisone, prednisolone, methylprednisolone, triamcinolone, deflazacort, betamethasone and dexamethasone.

17. The method of claim 15, wherein the subject is suffering from septic shock.

18. The method of claim 15, wherein the subject is suffering from Crohn's disease.

19. The method of claim 15, wherein the subject is suffering from asthma.

20. The method of claim 15, wherein the subject is suffering from graft versus host disease or transplant rejection.

21. The method of claim 15, wherein the subject is suffering from an autoimmune disease or disorder.

22. The method of claim 15, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of asthma, adult respiratory distress syndrome, systemic

lupus erythematosus, inflammatory bowel disease, Crohn's disease, ulcerative colitis, multiple sclerosis, insulin-dependent diabetes mellitus, autoimmune arthritis, **rheumatoid arthritis** , juvenile **rheumatoid arthritis**, psoriatic arthritis, inflammatory pulmonary syndrome, pemphigus vulgaris, idiopathic thrombocytopenic purpura, autoimmune meningitis, myasthenia gravis, autoimmune thyroiditis, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, Sjogren's Syndrome, keratoconjunctivitis sicca secondary to Sjogren's Syndrome, alopecia areata, allergic responses due to arthropod bite reactions, aphthous ulcer, iritis, conjunctivitis, keratoconjunctivitis, cutaneous lupus erythematosus, scleroderma, vaginitis, proctitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, erythema nodosum leprosum, autoimmune uveitis, allergic encephalomyelitis, aplastic anemia, pure red cell anemia, idiopathic thrombocytopenia, polychondritis, Wegener's granulomatosis, chronic active hepatitis, Graves ophthalmopathy, primary biliary cirrhosis, uveitis posterior and interstitial lung fibrosis.

23. The method of claim 15, wherein the subject is suffering from an acute inflammatory disorder.

24. The method of claim 15, wherein the subject is suffering from a chronic inflammatory disorder.

25. The method of claim 24, wherein the ICE inhibitor and the corticosteroid are administered such that steroid resistance in the subject is reversed, as compared to when a corticosteroid alone is administered to the subject.

26. The method of claim 24, wherein the ICE inhibitor and the corticosteroid are administered such that steroid sensitivity in the subject is increased, as compared to when a corticosteroid alone is administered to the subject.

27. The method of claim 24, wherein the ICE inhibitor and the corticosteroid are administered to the subject according to a schedule that reduces the dosage of the corticosteroid over time and the method ameliorates a steroid rebound effect associated with administration of reduced dosages of the corticosteroid.

28. A method for modulating responsiveness to a corticosteroid in a subject, comprising: selecting a subject in need of modulation of responsiveness to a corticosteroid, wherein the subject suffers from a condition normally responsive to corticosteroid therapy; and administering to the subject an interleukin-1 .beta. converting enzyme (ICE) inhibitor which antagonizes a factor that regulates production of interferon (IFN-.gamma.) in the subject, the ICE inhibitor being administered at a dosage and by a route sufficient to inhibit production of IFN-.gamma. in the subject, such that responsiveness of the subject to a corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.

29. The method of claim 28, wherein the subject is resistant to a corticosteroid prior to administration of the ICE inhibitor.

30. The method of claim 28, wherein the subject is responsive to a corticosteroid prior to administration of the ICE inhibitor but exhibits increased sensitivity to the corticosteroid after administration of the ICE inhibitor.

31. The method of claim 28, wherein treatment of the subject with a

corticosteroid is to be stopped and administration of the ICE inhibitor ameliorates a steroid rebound effect in the subject.

32. The method of claim 28, wherein the ICE inhibitor is an IFN-.gamma. inducing factor (IGIF) **antagonist**, the ICF inhibitor being administered at a dosage and by a route sufficient to inhibit IGIF activity in the subject.

33. A method for modulating responsiveness to corticosteroids in a subject comprising administering to the subject suffering from a condition normally responsive to corticosteroid therapy: an interleukin-1.beta. converting enzyme (ICE) inhibitor compound having the structure of Formula I: ##STR6## wherein R.sup.1 is hydrogen, C.sub.1 -C.sub.6 alkyl, or benzyl; R.sup.2 is --CHO, --COR.sup.a, or --CN; each R.sup.a is independently hydrogen or C.sub.1 -C.sub.6 alkyl; X is a bond, CH.sub.2, CHR.sup.5, NH, NR.sup.5, or O; R.sup.3 is aryl, substituted-aryl, heteroaryl, substituted-heteroaryl, cycloalkyl, substituted-cycloalkyl, heterocycle, or substituted-heterocycle; Y is absent, NR.sup.5, CO, S, O, SO.sub.2, --O(CHR.sup.5).sub.n --, CHR.sup.5, NR.sup.5 CO, NC(O)R.sup.5, CONR.sup.5, OCHR.sup.5, CHR.sup.5 O, SCHR.sup.5, CHR.sup.5 S, SO.sub.2 NR.sup.5, C.sub.1 -C.sub.6 alkyl, NR.sup.5 SO.sub.2, CH.sub.2 CHR.sup.5, CHR.sup.5 CH.sub.2, COCH.sub.2, or CH.sub.2 CO; R.sup.4 is absent, aryl, substituted-aryl, C.sub.1 -C.sub.8 alkyl, heteroaryl, substituted-heteroaryl, cycloalkyl, C.sub.1 -C.sub.6 alkyl, substituted-cycloalkyl, heterocycloalkyl, or substituted-heterocycloalkyl; each R.sup.5 is independently hydrogen, C.sub.1 -C.sub.6 alkyl, aryl, --(CH.sub.2).sub.n aryl, or --(CH.sub.2).sub.n cycloalkyl; each n is independently 0 to 5, m is 1 or 2, and the pharmaceutically acceptable salts, esters, amides, and prodrugs thereof; and a corticosteroid, such that responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.

34. A method for modulating responsiveness to a corticosteroid in a subject, comprising: selecting a subject in need of modulation of responsiveness to a corticosteroid, wherein the subject suffers from a condition normally responsive to corticosteroid therapy; and administering to the subject an interleukin-1.beta. converting enzyme (ICE) inhibitor compound having The structure of Formula I: ##STR7## wherein R.sup.1 is hydrogen, C.sub.1 -C.sub.6 alkyl, or benzyl; R.sup.2 is --CHO, --COR.sup.a, or --CN; each R.sup.a is independently hydrogen or C.sub.1 -C.sub.6 alkyl; X is a bond, CH.sub.2, CHR.sup.5, NH, NR.sup.5, or O; R.sup.3 is aryl, substituted-aryl, heteroaryl, substituted-heteroaryl, cycloalkyl, substituted-cycloalkyl, heterocycle, or substituted-heterocycle; Y is absent, NR.sup.5, CO, S, O, SO.sub.2, --O(CHR.sup.5).sub.n --, CHR5, NR.sup.5 CO, NC(O)R.sup.5, CONR.sup.5, OCHR.sup.5, CHR.sup.5 O, SCHR.sup.5, CHR.sup.5 S, SO.sub.2 NR.sup.5, C.sub.1 -C.sub.6 alkyl, NR.sup.5 SO.sub.2, CH.sub.2 CHR.sup.5, CHR.sup.5 CH.sub.2, COCH.sub.2, or CH.sub.2 CO; R.sup.4 is absent, aryl, substituted-aryl, C.sub.1 -C.sub.8 alkyl, heteroaryl, substituted-heteroaryl, cycloalkyl, C.sub.1 -C.sub.6 alkyl, substituted-cycloalkyl, heterocycloalkyl, or substituted-heterocycloalkyl; each R.sup.5 is independently hydrogen, C.sub.1 -C.sub.6 alkyl, aryl, --(CH.sub.2).sub.n aryl, or --(CH.sub.2).sub.n cycloalkyl; each n is independently 0 to 5, m is 1 or 2, and the pharmaceutically acceptable salts, esters, amides, and prodrugs thereof, the compound being administered at a dosage and by a route sufficient to inhibit production of IFN-.gamma. in the subject, such that responsiveness of the subject to a corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.

35. A method of claim 9, wherein the subject is suffering from an

immunoinflammatory disease or disorder selected from the group consisting of pemphigus vulgaris, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, alopecia areata, allergic responses due to arthropod bite reactions, cutaneous lupus erythematosus, scleroderma, vaginitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, and erythema nodosum leprosum.

36. A method of claim 9, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of multiple sclerosis, autoimmune arthritis, **rheumatoid arthritis**, juvenile **rheumatoid arthritis**, psoriatic arthritis, autoimmune meningitis, myasthenia gravis and allergic encephalomyelitis.

37. A method of claim 9, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of systemic lupus erythematosus, inflammatory bowel disease, Crohn's disease, ulcerative colitis, insulin-dependent diabetes mellitus, aphthous ulcer, proctitis, Wegener's granulomatosis, chronic active hepatitis, and primary biliary cirrhosis.

38. A method of claim 9, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of iritis, conjunctivitis, keratoconjunctivitis, autoimmune uveitis, Graves ophthalmopathy, and uveitis posterior.

39. A method of claim 9, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of idiopathic thrombocytopenic purpura, autoimmune thyroiditis, Sjogren's Syndrome, keratoconjunctivitis sicca secondary to Sjogren's Syndrome, aplastic anemia, pure red cell anemia, idiopathic thrombocytopenia, and polychondritis.

40. The method of claim 9, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of asthma, adult respiratory distress syndrome, inflammatory pulmonary syndrome, and interstitial lung fibrosis.

41. A method of claim 22, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of pemphigus vulgaris, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, alopecia areata, allergic responses due to arthropod bite reactions, cutaneous lupus erythematosus, scleroderma, vaginitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, and erythema nodosum leprosum.

42. A method of claim 22, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of mutiple sclerosis, autoimmune arthritis, **rheumatoid arthritis**, juvenile **rheumatoid arthritis**, psoriatic arthritis, autoimmune meningitis, myasthenia gravis and allergic encephalomyelitis.

43. A method of claim 22, wherein the subject is suffering from an innumoinflammatory disease or disorder selected from the group consisting of systemic lupus erythematosus, inflammatory bowel disease, Crohn's disease, ulcerative colitis, insulin-dependent diabetes mellitus, aphthous ulcer, procitis, Wegener's granulomatosis, chronic active hepatitis, and primary biliary cirrhosis.

44. A method of claim 22, wherein the subject is suffering from an inflammatory disease or disorder selected from the group consisting of

ireitis, conjunctivitis, keratoconjunctivitis, autoimmune eveitis, Graves ophthalmopathy, and uveitis posterior.

45. A method of claim 22, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisiting of idiopathic thrombocytopenic purpura, autoimmune thyroiditis, Sjogren's Syndrome, keratoconjunctivitis sicca secondary to Sjogren's Syndrome, aplastic anemia, pure red cell anemia, idiopathic thrombocytopenia, and polychondritis.

46. The method of claim 22, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of asthma, adult respiratory distress syndrome, inflammatory pulmonary syndrome, and interstitial lung fibrosis.

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CLM What is claimed is:

1. An **antibody** directed against a interleukin-12 (**IL -12**) beta2 receptor protein which protein (a) has low binding affinity for human **IL-12**, and (b) when complexed with a human **IL-12** beta1 receptor protein forms a complex having high binding affinity to human **IL-12**.